EFFECT OF ALKALINIZATION OF LIGNOCAINE HYDROCHLORIDE ON BRACHIAL PLEXUS BLOCK

THESIS
FOR
DOCTOR OF MEDICINE

(Anaesthesiology & Critical Care)

M.L.B. MEDICAL COLLEGE
BUNDELKHAND UNIVERSITY,
JHANSI (U.P.)

2002

RUBY MEHTA

DEPARTMENT OF ANAESTHESIOLOGY & CRITICAL CARE
M.L.B. Medical College
Jhansi.

CERTIFICATE

This is to certify that the work entitled "To study the effect of alkalinization of Lignocaine hydrochloride on Brachial plexus block". Which is being submitted as a thesis for M.D. (Anaesthesiology) examination 2002 of Bundelkhand University by Dr. Ruby Mehta has been carried out in the Department of Anaesthesiology & critical care, M.L.B. Medical College, Jhansi.

She has put in the necessary stay in the department as per university regulations.

Dated: 28.2.02

(Dr. A.K Gurwara)
M.S., D.A.
Professor and Head
Department of Anaesthesiology
& Critical Care
M.L.B. Medical College
Jhansi.

DEPARTMENT OF ANAESTHESIOLOGY & CRITICAL CARE
M.L.B. Medical College
Jhansi.

CERTIFICATE

This is to certify that the work entitled "To study the effect of alkalinization of Lignocaine hydrochloride on Brachial plexus block" has been carried out by Dr. Ruby Mehta under my direct supervision and guidance. The techniques and statistical methods used in this thesis have been undertaken by the candidate herself and checked by me from time to time.

Dated: 28.2.02

(Dr. D.D. Verma)
M.D., D.A.
Professor
Department of Anaesthesiology
& Critical Care
M.L.B. Medical College
Jhansi.

(Guide)

DEPARTMENT OF ANAESTHESIOLOGY & CRITICAL CARE
M.L.B. Medical College
Jhansi.

CERTIFICATE

This is to certify that the work entitled "To study the effect of alkalinization of Lignocaine hydrochloride on Brachial plexus block" has been carried out by Dr. Ruby Mehta under my direct supervision and guidance. The techniques and statistical methods used in this thesis have been undertaken by the candidate herself and checked by me from time to time.

Dated: 28.02.2K+2

(Dr. (Mrs) Veena Gupta)
M.D., D.A.
Associate Professor
Department of Anaesthesiology
& Critical Care
M.L.B. Medical College
Jhansi.
(Co-Guide)

ACKNOWLEDGEMENT

I am at loss of words when I wish to express the feeling of gratitude I have towards all who helped me in this work.

"The path to reason and knowledge is difficult but it is the road to tread and those who dare succeed". This is what has been inculcated to our growing intellect by our honourable Professor & Head Dr. A.K. Gurwara M.S., D.A. Department of Anaesthesiology & Critical care M.L.B. Medical college Jhansi. His sense of precision, unflinching tenacity, passion for reason, deep knowledge and experience was a constant source of inspiration to me.

Words and lexicons connot do full justice in expressing my reverence and profound gratitude to Dr. D.D. Verma M.D., D.A. Professor, Department of Anaesthesiology & Critical care, M.L.B. Medical college, Jhansi my Guide under whose benevolence and able guidance, I read, learned and ventured to write. Still I take this opportunity to acknowledge most humbly from the inner recess of my heart and with a deep sense of gratitude my indebtness to him. His inspiring benefaction bestowed upon me so generously, helped me to carry out this present work.

Gratitude has no means of expression but at the core of my heart, there is profound feeling of regard and thankfulness for my co-guide Dr. (Mrs) Veena Gupta M.D., D.A. Associate Professor, Department of Anaesthesiology and Critical care, M.L.B. Medical college, Jhansi whose invaluable guidance helped me to complete my work. With sincere thanks, I acknowledge her affectionate nature and constant encouragement that she has rendered to me during my study.

I wish to express my heart felt gratitude to Dr. P. Sahi M.D, D.A. Associate Professor, Department of Anaesthesiology & Critical care, M.L.B. Medical college, Jhansi for his generosity, loving attitude, practical approach to life which would always act as a pointer in my life. I thank all my teachers.

I extend my special thanks to Dr. Deepika Jain and Dr. Neerja Arora who have helped me tide over difficult situations faced during my study.

All my colleagues and juniors in the Department of Anaesthesiology deserve my heartiest thanks for their co-operation and support during the course of this work.

I also wish to thank Mr. Zaheer Hasan, clerk, Department of Anaesthesiology for his co-operation, without which this work would not have finished in time.

I feel highly obliged to my computer operator Mr. Rafat Siddiqui (Yes Computers) for preparing this manuscript in an exemplary manner.

Every particle of me is indebted to my parents and in-laws for their love, sacrifice, care and inspiration at every moment of my life. Last and certainly not the least, this study would not have been possible without the active participation of my husband Dr. Ravi Mohan Singh. He has always been available with ready invaluable suggestions and unending encouragement. This study carries imprints of his help, wise and meticulous advice I'am really thankful to him.

Ruby Mehta

Dated : 28.2.02

Dr. Ruby Mehta

Dedicated

to

my

Husband

CONTENT

Summary in separate cover .

INTRODUCTION

INTRODUCTION

"If a man, for instance, could have his hand so obtunded that he could see, but not feel, the performance of amputation upon his own fingers, the practice of anaesthesia in surgery world, in all likelihood, advance and progress even still more rapidly than it has done".

This striking appreciation of the benefits of local anaesthesia was published in 1848 by James Young Simpson, decades before regional anaesthesia became a practical possibility.

A properly conducted regional technique is not only economical but it also provides good operative conditions and there is virtually no disturbance in various organ system, this is of particular importance in patients with significant medical problems and those arriving in operation room with a full stomach. In such cases, regional anaesthesia allows surgery to be performed with the patient awake, co-operative minimizing the danger of aspiration as against general anaesthesia.

Many anaesthetist believe that regional anaesthesia has its greatest usefulness in surgery over extremities. Brachial plexus bock has become the most commonly used technique for anaesthesia of the upper limb. Brachial plexus block was first practiced by Prof. Halsted (1884) by directly exposing the nerve roots in the neck. Supraclavicular approach to brachial plexus described by Kulenkanff is the most popular approach among the various approaches available. The inherent advantage of this technique are :-

(a) Easy accessibility

(b) Simplicity of the technique

(c) Predictable landmarks

(d) Need for lesser volume of drugs and lastly it is performed for surgery over arm, forearm and hand.

These advantages coupled with the advantage offered by regional anaesthesia in comparison to general anaesthesia makes it a more applicable procedure. In contrast the role of regional anaesthesia in clinical practice is limited by the capabilities of the local anaesthetic drugs. Two practical disadvantages inherent with the agents currently available are, firstly the drugs takes a considerable time to work and secondly duration of anaesthesia is limited which may be inadequate for protracted operations.

Since long, lot of efforts have been made to overcome such problems in otherwise safe, ecnomical and cheap procedure.

Observations of different researchers led to the suggestion that relative alkalinity of the local anaesthetic agent can be a major determining factor in altering the onset and duration of block.

This inspired us to conduct the study to evaluate the effect of alkalinzation of lignocaine hydrochloride on brachial plexus block.

MATERIAL

&

METHOD

MATERIAL AND METHOD

The present study entitled ***"Effect of Alkalinization Of Lignocaine Hydrochloride On Brachial Plexus Block"*** was undertaken in the Department of Anaesthesiology and Critical care, M.L.B. Medical College and Hospital, Jhansi during the period of April 2000 to November 2001.

SELECTION OF CASES

The cases for this study were selected from the indoor patient of M.L.B. Medical College and Hospital, Jhansi. The age of the adult subject ranged form 20 to 60 years of either sex. Cases requiring upper limb surgery and who were suitable for supraclavicular brachial plexus block were enrolled. Prior to the selection of cases a thorough pre-anaesthetic clinical evaluation of all cases was carried out and only ASA grade I and II patients were included in this study.

Exclusion criteria's included

- Progressive neurological disorder.
- Severe kidney or liver dysfunction.
- History of adverse reaction to local anaesthetic drugs
- Patients with bilateral upper limb surgery or patients who required local anaesthetics on two different areas.
- Patients weighing less than 50 kilogram or more than 70 kilogram were also excluded from the study because we used a fixed amount of local anaesthetic.
- Opposite side pneumothorax/collapsed or partially collapsed lung.

- Any skin infection over local area.

PREPERATION OF CASES

After explaining the details of the procedure, an informed consent was taken from the case. No premedication was administered in the night before surgery or in the operation theatre.

On arrival in the operating theatre an intravenous line was established with 18 guage cannula inserted into peripheral vein for infusion of intravenous fluids.

STUDY DESIGN

The cases were randomly allocated into three groups of 20 cases each according to the drug received

Group I- Subjects were given 20 ml of 2% lignocaine hydrochloride with adrenaline (1: 200000), ($p^H = 3.21$)

Group II- Subjects received freshly prepared alkalinized 20ml solution of 2% lignocaine hydrochloride with adrenaline (1:200000), by adding 1ml of 7.5% (wt/vol) sodium bicarbonate. ($p^H = 6.21$)

Group III- Subjects received freshly prepared alkalinized 20ml solution of 2% lignocaine hydrochloride with adrenaline (1:200000), by adding 2ml of 7.5% (wt/vol) sodium bicarbonate. ($p^H = 6.67$)

Alkalinization :- Alkalinized solution was freshly prepared prior to injection by adding required amount of sodium bicarbonate 7.5% (wt/vol) to 20 ml of lignocaine hydrochloride with adrenaline solution. Mixture was inverted. without shaking, 30 times over a period of 45-60 sec. pH of the solution was estimated by an electronic pH meter.

TECHNIQUE OF SUPRACLAVICULAR BLOCK

Positioning :-

Patient was asked to lie supine with a pillow under the shoulders and the head turned away from the site of the injection. The affected arm kept by the side of the body, shoulders lowered by asking the patient to reach for his knees so that subclavian artery become easily palpable.

After taking all sterile precautions the site of injection was cleared with betadine and then with spirit and was allowed to dry. The sourrounding area was covered with sterile drapes.

Anatomical landmarks were palpated. Midclavicular point was taken and just above that subclavian artery was palpated. A skin wheal was raised with 1ml of 2% lignocaine hydrochloride 1cm above the midclavicular point, just lateral to subclavian artery avoiding the external juglar vein.

The patient was instructed not to move the arm, instead advised to say 'Yes' when he had tingling sensation in the upper limb.

Now a 23 guage needle about 5 cm long attached to a 20ml syringe containing the local anaesthetic solution was introduced through the skin wheal in a backward, inward and downward direction towards the upper surface of first rib over which plexus lies. When the patient said 'Yes' the needle was stopped then after checking for negative aspiration the local anaesthetic solution was injected slowly.

In cases where the tingling sensation was not elicited the needle was further introduced in the same direction mentioned earlier to meet the first rib. Once the first rib was met 'Rib walking' was done in the

anteroposterior direction to elicit tingling sensation. As soon as it was elicited, aspiration done and local anaesthetic solution was injected there.

If tingling sensation was not elicited even after rib walking then the needle direction was changed slightly and the procedure was repeated. Even with this if tingling sensation was not elicited then the drug was injected slowly as the needle was withdrawn towards the skin. This is called 'Patrick's Technique'. During the procedure if subclavian artery was punctured accidently the needle was withdrawn immediately and pressure was applied over the artery for some time and then procedure was repeated with a little change in the direction of the needle.

Once the drug was injected the pillow under the shoulder was removed and the head was now turned to the same side of injection, massaging was done over the site of injection. Onset of sensory and motor block were tested respectively at every one minute interval, for a maximum period of thirty five minutes from the time of starting drug injection.

PARAMETERS RECORDED

- **Onset of sensory block :-** Determined by no response to temperature (tested by swab soaked in spirit), fine touch, pin prick and deep pressure sensation.
- **Onset of motor block :-** Judged by paresis or paralysis. Onset time for paresis was taken from the time of injection to the loss of dorsiflexion of the wrist joint and paralysis from the time of injection to the loss of finger movements.
- **Quality of the block :-** Complete, Incomplete, Failed

Complete : When all of the parameters were acheived within thirty five minutes of duration from the time of injection.

Incomplete : When any of the parameters except pinprick could not be acheived within thirty five minutes of duration from the time of injection.

Failed : When even pinprick could not be blocked for duration more than thirty five minutes from the time of injection.

- **Duration of block**

Sensory : Time taken from the complete onset of sensory block till the patient respond to pinprick.

Motor : Time taken from the of loss of finger movements till the patient started moving his fingers.

MONITORING

Patients pulse rate, blood pressure, respiratory rate and oxygen saturation was monitored both preoperatively, 15 minutes, 45 minutes intra operatively after injection and postoperatively. Any sign and symptom for local anaesthetic toxicity was looked for at the time of injection, intra-operatively as well as post-operatively.

POST OPERATIVE

All cases were kept under observation for 24 hrs.

RE-VIEW
OF
LITERATURE

REVIEW OF LITERATURE

Benjamin ward Richardson, an eminent victorian experimented with electricity and cold water for producing local anaesthesia before he finally introduced ether spray which worked by evaporation and was the only practical method of local anaesthesia towards the end of 18th century. In 1855 wood, a physician called father-in-law of local anaesthesia first developed hypodermic syringes and needle to produce nerve block by drug injection. All he lacked was an agent which would work. The first ever local anaesthetic agent cocaine, was produced as pure white crystals by Niemann in 1860, but, was first used as local anaesthetic agent by Carlkoller, a young graduate of the Vienna Medical School for anaesthetizing cornea during eye surgery in 1884 and lead to the introduction of modern local analgesia. Absolute priorty for peripheral nerve block goes to Sir William Halsted in 1884 when he freed the cords and nerves of the brachial plexus after blocking the roots of the neck with cocaine solution. In 1887, Crile disarticulated the shoulder joint after making the arm insenstive by blocking the brachial plexus by direct intraneural injection of each nerve trunk with 0.5% cocaine under direct vision.

The major factor to abandon cocaine as a local anaesthetic agent was its toxicity, difficulty to sterlize, brief in duration and addiction properties. Later Schleich in Germany and Reclus in France developed safe dose regeimes for cocaine and popularized infilteration anaesthesia. Braun increased its duration and reduced toxicity by the use of a torniquet and later by adding adrenaline to the solution. Widespread use of local methods had to await the introduction of safer drugs. Amylocaine was introduced in 1903 but lost popularity due to irritant property. Einhoun in

1904 devloped Procaine which has low toxicity, lacks addiction properties but was still far from being ideal as it hydrolysis rapidly and may induce allergic reaction. By the first 50 years after Koller's introduction of cocaine, the only local anaesthetic agent to become established were amethocaine and cinchocaine. Both were potent and toxic.

The 1940 saw the start of the next major advance. Working in stockholm on the structure of the alkaloid gramine, Erdtman-an organic chemist tasted one of the substances that had been produced as a precusor of gramine. The significance of ensuing numbness was appreciated immediately and the search for a clinically useful derivative was started by Erdtman and continued by Nil's Lofgren who synthesized lignocaine in 1943. Lofgren in 1948 laid the foundation of subsequent studies of local anaesthetic drugs. From these studies have come derivatives of lignocaine such as mepivacaine, prilocaine bupivacaine and etidocaine. While the introduction of these agents has considerably widned the scope of local anaesthesia but unfortunately the years between 1950 and 1955 saw a sharp decrease in the use of local anaesthesia because of the fear of severe neurological damage and many advances in general anaesthesia taking place. But due to the efforts of anaesthetist it was not so. Various pharmacological as well as technical advances were advocated to increase the potency, quality and to reduce toxicity of the local anaesthetic agents.

Ritchie and Greengard (1965) suggested that it is the lipid soluble moiety of local anaesthetic agent more concerned with the penetration of tissue barriers, and that the electrically charged cation form probably is the active agent that finally engages at the charged surface of an excitable membrane.

Bromage PR et al (1967) demonstrated improved quality of block, when carbon dioxide enriched local anaesthetic was injected epidurally and concluded this not due to greater concentration of anaesthetic cation at the nerve axon but also due to direct stabilizing effect of carbon dioxide on excitable tissues.

N Harley, J Gjessing (1969) compared the effect of adding either adrenaline or hyaluronidase or both of these drugs to 1% mepivacaine for brachial plexus block through supraclavicular approach. It was found that addition of adrenaline apparently increased the duration of sensory block while with hyaluronidase decreased intensity of block was noticed and with both adrenaline and hyaluronidase no effect was appreciated.

Bromage PR. et al (1972) sought for the latency and duration of supraclavicular brachial block in 183 patients using carbonated lignocaine and bupivacaine hydrochloride. He found that carbonated lignocaine had the shortest latency and shortest duration. Bupivacaine had longest latency and longest duration, while mixture of both carbonated lignocaine and bupivacaine hydrochloride produced rapid onset with moderately longer duration.

Gray Strichartz (1976) concluded that local anaesthetic block nerve conduction by preventing the increase in membrane permeability to sodium ions and it is the cationic protonated form that appears to be more active than the neutral form.

Alon. P. Winnie, Cheng-Hin Tay et al (1977) described a new model for the study of pharmacokinetics of local anaesthetic which allows the seperate determination of onset and recovery of sensory and motor block in peripheral (mantle) and central (Core) bundles within the nerve

trunks. In his study he concluded that motor fibres located peripherally blocked first and were last one to recover in comparison to sensory fibres situated centrally.

Daniel C. Moore (1981) reported the pH values of the commonly used, commercially prepared local anaesthetic agent with and without epinephrine 1: 200,000 as well as additives that they contain by the Beckman model 3560 digital pH meter. With the exception of chlorprocaine, all solutions of the local anaesthetic drugs without epinephrine had pH values of 4 or greater and solution with epinephrine had pH values less than 4. pH of epinephrine was 3.3. Addition of additives further reduced the pH. In his study he specified 2% lignocaine without epinephrine having pH of 6.32, while commercially prepared 2% lignocaine having pH of 3.86.

The structure and the physiochemical properties of local anaesthetics were reviewed by ***Courtney and Strichartz (1987)***. They reported that hydrophobicity increases both potency and duration while molecular size influences dissociation of local anaesthetics from their receptor site.

Radha Sukhani and Alon. P. Winnic (1987) experimented on fifty healthy patients undergoing upper extremity surgery by subclavian perivascular technique. They compared carbonated lignocaine and lignocaine hydrochloride and found that the carbonated lignocaine reduced the latency of anaesthesia by 45% as compared with its hydrochloride salt, producing complete motor block in almost as many as twice patients. The duration of anaesthesia provided by the two agents was virtually identical as was duration of motor block. They concluded this due to the liberation of carbon dioxide from the carbonated solution which diffuses very rapidly across a nerve membrane causing a fall in the intracellular pH, and the

production of cationic trap which results in marked increase in the amount of active cation available at the receptors sites on the sodium channels inside the nerve membrane. Furthermore carbon dioxide may also have a direct stabilizing effect on the nerve membranes.

Paula M. Bokesch et al (1987) indicated in their experiment that carbondioxide potentiates conduction block with lignocaine either by a direct effect on the membrane or by its indirect action on intracellular pH.

R. Martin, L. Beauregard, Y. Lasnarcha, JP Tetrault (1987) in a study on axillary block found that the lignocaine hydrochloride and lignocaine hydrocarbonate both gave shorter latency of analgesia than mepivacaine. Duration of analgesia, quality of sensory and motor block were not found to be statistically different between these groups.

MD Bedder, R Kozody, DB Craig (1988) studied the effect of alkalinization of bupivacaine 0.5% by subclavian perivascular brachial plexus block in 60 patients. He concluded that the alkalinization of bupivacaine 0.5% solution does not confer any added clinical advantage.

Christian Verborgh, Maric Anne clarys and Frederic Camu (1991) in their double blind study on forty patients investigated the effect of adding 1.4% bicarbonate to 0.5% bupivacaine on latency of sensory and motor block after epidural administration. They concluded that alkalinization of 0.5% bupivacaine offers no improvement in the onset of epidural block.

Joseph J. Quinlan, Karole Oleksey and Frank L. Murphy (1992) examined the onset and distribution of sensory and motor block, along with venous mepivacaine concentrations after axillary block with 1.25% mepivacaine with and without bicarbonate. There was no statistically

significant differences between the alkalinized and placebo groups with respect to the distribution of analgesia or anaesthesia, time to the onset of analgesia or time to the onset of paresis. However, alkalinization significantly decreased the time to the onset of anaesthesia as well as paralysis in the medial cutaneous nerve of forearm, the median nerve and the ulnar nerve.

Roberts JE, Macleod BA and Hollanods RH. (1993) carried out a double blind study to determine the effect of pH and the addition of hyaluronidase to a mixture of lignocaine and bupivacaine on the efficacy of peribulbar anaesthesia. The solution containing hyaluronidase with pH adjusted to 6.7 by alkalinization was found to be most effective. Presence of hyaluronidase without alkalinization did not improve the efficacy and similarly, alkalinization in the absence of hyaluronidase was ineffective.

AV Capogna G, Celleno D, Laudano D and Giunta F. (1995) determined the efficacy of alkalinization using different local anaesthetic solutions and different regional blocks over 180 patients in a randomized, double blind fashion. The local anaesthetic solutions studied were bupivacaine, mepivacaine and lignocaine; the regional blocks studied were epidural block, axillary brachial plexus block, femoral and sciatic nerve block. He came to the conclusion that alkalinization produced the best results with lignocaine and bupivacaine for epidural block, with lignocaine for brachial plexus block, and with mepivacaine for sciatic and femoral nerve blocks.

AV Gormley WP, Hill DA, Murray JM and Fee JF (1996) observed the effect of alkalinization of lignocaine on axillary brachial plexus block. Forty two patients scheduled for upper limb surgery received axillary brachial plexus anaesthesia using a cannula technique. Patients were

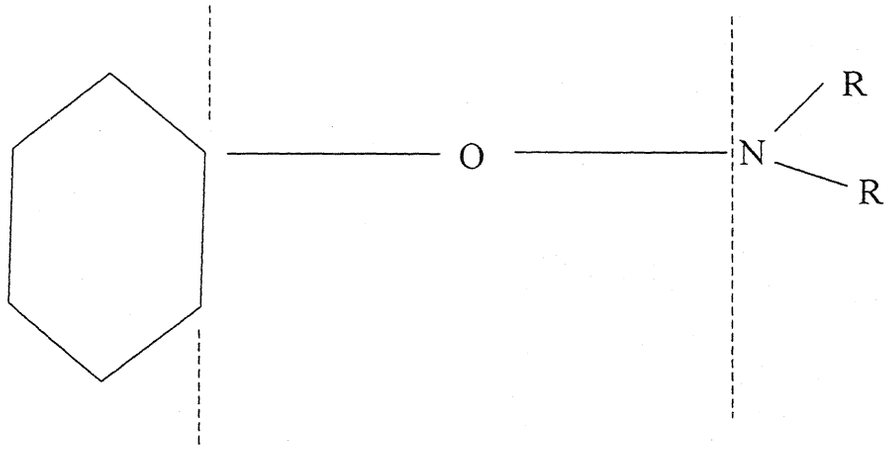
randomly allocated to receive either commercially prepared lignocaine 1.5% with adrenaline (1:200,000), (p^H 4.2) or lignocaine 1.5% with freshly added adrenaline (1:200,000) (p^H 7.2). No significant difference was seen in the incidence of satisfactory block or distribution of anaesthesia between the two groups. The percentage of patients with complete anaesthesia at 10, 20 and 30 minutes following injection was significantly increased in the alkalinized group with regard to the ulnar nerve, median nerve, and the median cutaneous nerve of the arm. In the alkalinized group, there was a significant reduction in the time of attainment of useful anaesthesia and a reduced requirement for adjuvants.

Mack YH Chow, Alex TH Sia, CK koay and YW Chan (1998) assessed the onset of sensory and motor block as well as the distribution of sensory block after axillary brachial plexus block. 1.5% lignocaine hydrochloride containing adrenaline (1:200,000) with and without sodium bicarbonate was given in 38 patients and they found that alkalinization of lignocaine did not offer any significant clinical advantage in axillary brachial plexus block.

Ririe DG,Walker FO, James RL, and Butterworth J (2000) performed median nerve blocks in 10 volunteers in a randomized, double blind cross over study to compare the effects of 1% plain lignocaine with 1% lignocaine mixed with sodium bicarbonate 0.1 mmol/liter. Sensations of hot, cold, pin prick, light touch and motor sensations were assessed at two minutes intervals. pH was 6.4±0.1 for plain lignocaine and 7.7±0.2 for alkalinized lignocaine. The final data suggested that addition of bicarbonate to lignocaine for median nerve block significantly increased the rate of motor block without changing the onset or extent of sensory block.

PHARMACOLOGY OF LOCAL ANAESTHETIC DRUGS

A local anaesthetic is a drug which reversibly blocks the transmission of peripheral nerve impulses. These agents conform to a common structural arrangement consisting of a benzene ring attached to an amine group by an intermediate chain, which includes either an ester or an amide linkage



Aromatic Group | Intermediate Chain | Amine Group

Mechanism of Action:-

During the resting phase the interior of a peripheral nerve axon has a potential difference of about –70 mv. relative to the outside. The resting potential exists because there are more anions than cations within the cell. In the present context the most important ions are sodium and potassium. The high extracellular sodium concentration is maintained because at rest the membrane is impermeable to sodium. However it is freely permeable to potassium ions, which diffuse out of the cell until the negative intracellular electrochemical potential created by their loss balances the concentration gradient.

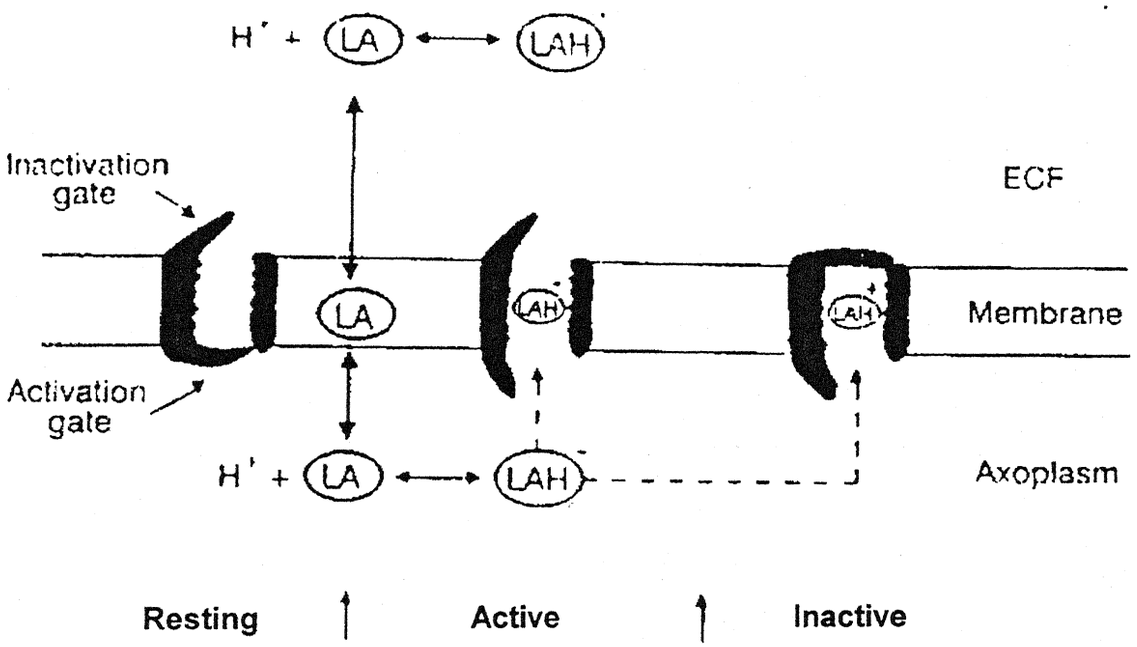


Fig 1 : Mechanism of Sodium channel inactivation by local anaesthetic

LA - Local Anaesthetic Base
LA H - Ionised local Anaesthetic
ECF - Extra cellular fluid

Depolarization of the fibre is the result of a sudden increase in membrane permeability to sodium. Sodium ions enters as a result of changes in the configuration of large protein molecules present in the cell membrane. Nerve stimulation causes "channels" in these proteins to open and it is through these channels that sodium ions enter the axoplasm. Entry of the positively charged sodium ions raises the membrane potential to about +20mv, at this point the electrochemical and concentration gradients for sodium balances one another and the channel close. Both concentration and electrochemical gradients then favours movement of potassium out through the membrane until the resting potential is restored. The impulse generated is transmitted along the axon because a local current flows between the depolarized segment of nerve and the next segment. The voltage change associated with this current opens the sodium channels in the next section, so that the action potential is propogated along the nerve.

Local anaesthetics are usually injected as acid solutions of the hydrochloride salt (pH <5). In this form the amine group is ionized and the drug becomes soluble in water and therefore suitable for injection. After injection tissue buffering raises the pH and a percentage of the drug dissociates to become free bases, the amount depending on the "dissociation constant" of the indivual drug. Being lipid soluble, the free base is able to penetrate both the nerve coverings and the lipid cell membrane to reach the interior of the axon where a portion re-ionizes. The re-ionized portion then enters the sodium channels and may be thought of as simply "plugging" them so that sodium ions cannot enter the cell. As a result, action potential are neither generated nor propogated and conduction block occurs.

Narahashi and Frazier (1971) suggested that the site at which local anaesthetics act, at least in their charged form, is accessible only from the inner surface of the membrane.

Retchie and Strichartz (1987) demonstrated conduction block in squid giant axons form which axoplasm has been removed and proved that conduction block occurs by decreasing or preventing the large transient increase in the permeability of excitable membrane to sodium ions that normally is produced by a slight depolarization of the membrane.

Butterworth and Strichartz (1990) proved that the major mechanism of action of these drugs involves their interactions with one or more specific binding sites within the sodium channel. In ***1992 Catterall*** studied the structure and function of the sodium channel and other voltage gated ion channels.

VARIOUS OTHER THEORIES ON MECHANISM OF ACTION OF LOCAL ANAESTHETICS

1. **Membrane Expansion theory :** ***Seeman P. (1970)*** gave the membrane expansion theory of anaesthesia that the anaesthetics absorb to the hydrophobic regions of excitable membrane, expands some critical regions in the membrane thus preventing sodium permeability.
2. **Changes in Membrane surface charge theory:** ***Mc Laughlin S and Feinstein MB (1975)*** explained that the electrical surface potential will be altered by charged anaesthetics adsorbing to the membrane and thus affecting general membrane structure, thereby interfering with the normal operation of sodium channels.
3. **Specific Receptor Hypothesis :** ***Strichartz GR (1973)*** proposed third hypothesis that the local anaesthetics act by complexing with

specific receptors in the nerve membrane. The action of the drug is direct, not mediated by some change in general membrane properties. The idea of specific receptors was developed to explain the effects of intra-axonal quaternary lignocaine derivaties upon sodium permeability.

CARBONATED LOCAL ANAESTHETIC SALTS :

It has been known for a long time that the hydrogen ion concentration is an important factor in the uptake of local analgesic agents. Begnon in 1892 mentioned cocaine with alkali added, and in 1910 Gross suggested how alkalinized solutions worked. The theory of carbonated local analgesics has been ably put forward by Bromage in 1965.

Carbonated solution, like lignocaine carbonate, have a relatively high pH of 6.5 and are thus less demanding for the buffering capacity of the tissues (pH 7.4) than the commonly available local analgesics. On injection of this carbonated solution the free base is liberated, carbon dioxide rapidly diffuses into the axon interior and here the pH falls, which forces dissociation of the local anaesthetic to the cationic active form. This effect results in "ion-trapping" similar to that seen in the kidney, further favouring the rapid movement of the local anaesthetic into the axon.

In support of this theory it has been shown in vitro by Catchlove R.F.H. (1972) that the exposure of axons to equal amounts of carbonate or hydrochloride lignocaine resulted in a tenfold increase in the degree of block for the carbonate salt compared to the hydrochloride salt. The effect of carbon dioxide on local anaesthetic action in vitro depends on three mechanisms.

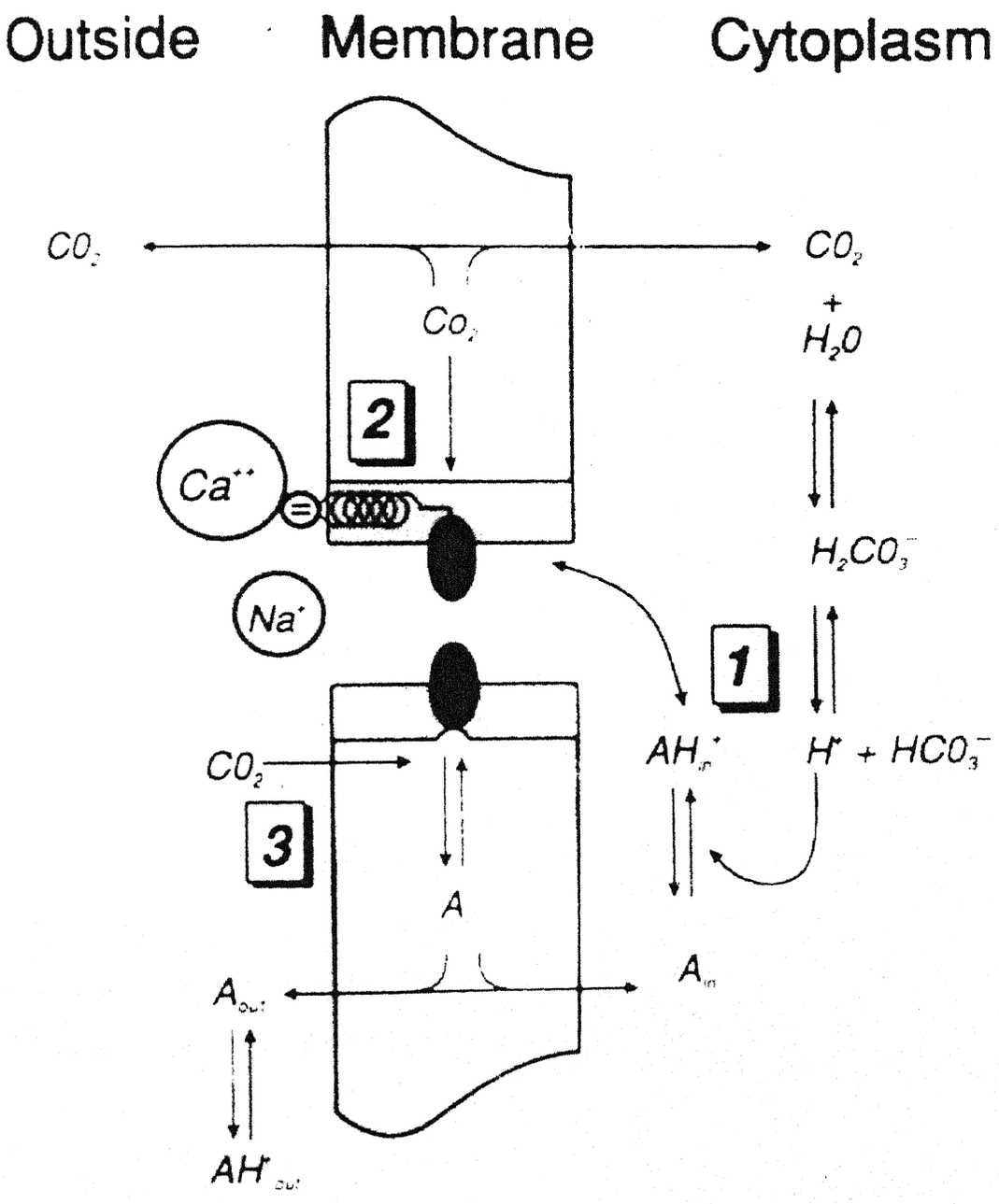


Fig 2 : Effect of carbon dioxide on local anaesthetic action

1. *Diffusion trapping*
2. *Direct Depressant effect of Co_2*
3. *Increase in sodium channel binding by local anaesthetic*

(a) Axoplasmic acidification with ion trapping of local anaesthetics.

(b) A general reduction in the safety margin for nerve impulse conduction induced by carbon dioxide.

(c) Species – specific increases in sodium channel binding by local anaesthetic.

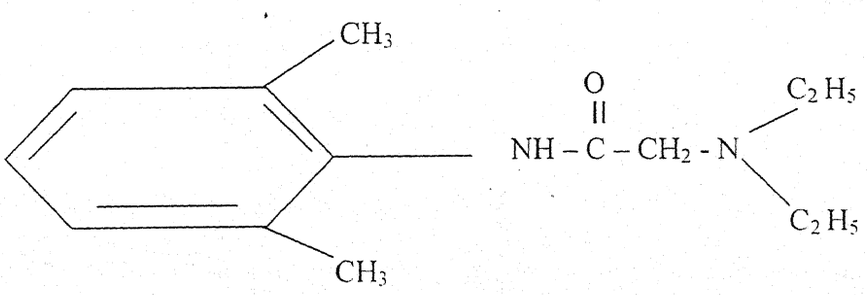
CLASSIFICATION OF LOCAL ANAESTHETIC AGENT :

ESTERS	***AMIDES***
➢ Cocaine	➢ Cinchocaine
➢ Benzocaine	➢ Lignocaine
➢ Procaine	➢ Mepivacaine
➢ Amethocaine	➢ Prilocaine
➢ Chloroprocaine	➢ Bupivacaine
	➢ Etidocaine
	➢ Ropivacaine

LIGNOCAINE

Lignocaine was synthesized in 1943 by Lafgren and Landquist and was first introduced in the clinical practice by Gordh in 1948. It is now one of the most widely used local anaesthetic agent. It's chemical name is – Diethylamino-2, 6-aceto-xylidine

It belongs to Anilide group of amide local anaesthetics



LIGNOCAINE

Physiochemical Properties

Lignocaine hydrochloride is an odourless, white crystalline powder. Its partition co-efficient is 2.9, pKa 7.9, molecular weight 234 and protein binding of 64%.

Solubility

It is freely soluble in water.

Stability

Very stable, the stability of lignocaine is absolute within a wide range of temperature and pH.

Sterlization

Vials containing lignocaine solution can be sterlized by boiling or autoclaving. Lignocaine crystals can be autoclaved for 6 hrs or subjected to multiple autoclaving without loss of potency.

Absorption Metabolism and Excretion

Lignocaine is absorbed rapidly after parentral administration from the gastrointestinal and respiratory tracts. Although it is effective when used without any vasoconstrictor, in the presence of epinephrine the rate of absorption and toxicity is decreased, and the duration of action usually is prolonged.

The highest concentration of the drug is found in kidney after systemic absorption. Appreciable levels are found in lungs spleen, heart and brain, rather low levels are found in liver.

The anaesthetic has high affinity for the fatty tissues. Lignocaine has a half life of 1.6 hrs. Most of the drug is metabolized by biotransformation into free and conjugated phenol. The ring structure is hydroxylated. It is

metabolized in the liver by enzyme oxidase to monoethylglycine and xylidide. The latter compound retains significant local anaesthetic and toxic activity. In man about 75% xylidide is excreted in the urine as 4-Hydroxy 2-6 diethyl aniline and less than 3% is excreted unchanged in urine.

Anaesthetic Properties

Lignocaine is useful for infilteration and block anaesthesia. It is effective topically when applied to mucous membrane and is a extremely valuable drug for regional block. Lignocaine is commonly made up with adrenaline in an attempt to delay the rate of absorption from the site of injection by producing local vasoconstriction. Commercially prepared solution should be injected immediately following its removal from the vial. The concentration which are adequate to produce analgesia for skin incision with lignocaine are.

For

Infilteration	} 0.5%
Intravenous regional block	
Minor nerve block	1%
Brachial plexus block	} 1.0% - 2%
Sciatic/Femoral block	
Epidural	1.5% - 2%
Spinal	2-5%

Greater concentrations than these may be used to produce more profound blocks of faster onset. The volume to be injected will depend on the particular technique.

Dosages :-

Basically it should be remembered that smallest dose producing desired result should be given. Dosage for deblitated and aged patients should be appropriately reduced. For children smaller amount of lignocaine in low concentration should be administered depending on body weight. The suggested maximum dose of lignocaine with adrenaline is 7mg/kg body weight and without adrenaline is 3 mg/kg body weight.

Onset of Anaesthesia :-

It occurs in approximately 5 minutes for minor nerve block and 5-20 minutes with major nerve block. The carbon dioxide salt of lignocaine has a more rapid onset time than the plain lignocaine and will produce brachial plexus block in approximately 8 minutes. Bromage PR and Robson JG (1970)

Duration of Anaesthesia :-

For minor and major nerve block duration of anaesthesia without epinephrine is 60-90 minutes and with epinephrine is 120-300 minutes Bromage PR and Robson JG. (1970)

Safe Dosage :-

Safe dose without epinephrine is 200 to 400 mg and with epinephrine it is 500 mg, regardless of the block, technique and the concentration, as long as the concentration is in the range of 0.5-2%.

Systemic Effects :-

a) **Effect on Central Nervous system :** Lignocaine causes initial sleepiness in many patients. It has also been used as an anticonvulsant in the treatment of status epilepticus.

b) **Effect on Cardiovascular System :** Lignocaine is a useful drug in the treatment of cardiac dysrrhythmias. It stablizes the membranes of damaged and excitable cells tending to supress ectopic foci. In therapeutic doses it causes no consistent change in the heart rate and does not depress conduction in purkinje fibers. Bigger J.T and Heisenbulted RH. (1969).

There is usually no myocardial depression, instead improvement in the cardiac output and blood pressure has been observed when used in cardiac dysrrhythmias (Harrison DC et al. 1963) The great value of lignocaine is in the acute treatment of ventricular dysrrhythmias after myocardial infarction or cardiac surgery. It has been seen that at a blood levels of approximately 3 μg/ml of lignocaine myocardial function is not greatly altered and may infact be improved. Jewitt D and Julion DC (1971)

c) **Neuromuscular function and ganglionic synapse**

Local anaesthetics also affect transmission at the neuromuscular junction. Similar effects occurs at autonomic ganglia. These effects are due to the block of the ion channel of the acetylcholine receptors. Neher and Steinbach (1978)

OTHER SIDE EFFECTS

Drug interactions

Lignocaine decreases the requirements of nitrous oxide and halothane in an additive fashion, i.e reduces halothane minimum alveolar concentration (MAC). Anaesthesia with nitrous oxide and halothane also decreases the clearance of lignocaine due to reduced liver blood flow and enzyme inhibition. Propranolol reduces the clearance of lignocaine while,

enzyme induction with barbiturates and phenytion increases the clearance of lignocaine. High concentrations of lignocaine depresses the twitch response at the neuromuscular junction and can prolong the action of non-depolarizing muscle relaxants.

Allergy

Many so called allergic reactions to these drugs are reactions to their additives and preservatives and not to the local anaesthetics - Methylparaben, a preservative, and sodium metabisulphite, an antioxidant, are the most common offenders. Reaction to ester drugs are more common and are due to a para-aminobenzoic acid metabolite in a manner somewhat similar to sulphonamide reactions.

LOCAL ANAESTHETIC TOXICITY

Toxic reactions are generally caused by

1. Relative overdose
2. Accidental intravascular injection leading to high plasma concentration.
3. Susceptibility of the individuals.
4. Various predisposing factors like liver disease, extremes of ages, pyrexia, shock, renal disease.

Clinical Feature Of Toxicity And Its Diagnosis

Local anaesthetic toxicity is a function of plasma free drug concentration and is influenced by the drug, the dose and the injection site. The spectrum of toxicity extends from mild and non-threatning to cardiorespiratory collapse and death.

The early symptoms of toxicity are numbness of the tounge and circumoral region. Light headedness and tinitus are encountered most frequently in the patients on intravenous antiarrhythmic therapy. They appear at plasma lignocaine concentration of about 5 μg/ml, the normal therapeutic range for artiarrhythmic activity being 2-4μg/ml. Further progression of toxic manifestation beyond mild central nervous system symptoms is an indication of impending seriousness. Drowsiness, visual disturbances and muscular twitching occurs at lignocaine concentration of 5-10 μg/ml. Above 10μg/ml convulsion coma and respiratory arrest are likely.

Serious central nervous system toxicity is indicative of imminent and potentially, more lethal cardiac toxicity, direct cardiovascular depression occurring at plasma lignocaine concentration greater than 20 μg/ml. Local anaesthetic directly depress myocardial conduction and contractility in a dose dependent manner. They bind to and inactivate myocardial sodium channels, reducing the velocity of cardiac action potential and prolonging the QRS interval. When plasma concentration rises towards toxic levels more and more sodium channels become inactivated until there is a generalised reduction in automaticity accompanied by negative ionotropy.

There may also be indirect effects of local anaesthetics on the heart mediated by the central nervous system. The early onset of life threatning ventricular arrhythmias is a prominent feature. Hypoxia, hypercarbia and acidosis will exacerbate local anaesthetic toxicity. The ensuing intracellular acidosis further promotes trapping of local anaesthetic cation. The heart is more resistant to the toxic effects of local anaesthetics than the brain. The plasma drug concentration required to produce cardiovascular collapse (CC) relative to central nervous system toxicity (CNS), the CC:CNS ratio is different for each agent. Lignocaine has ratio of 7:1

Prophylaxis and prevention of toxicity

The best prophylaxis for systemic local anaesthetic toxicity remains avoiding the administration of inappropriately large doses. Overdose can be avoided by a proper understanding of the pharmacology, especially the factors that affect absorption, distribution and elimination.

Inadvertent intravenous injection can often be avoided by careful technique and by allowing sufficient time for reflux of blood down the needle or catheter used for injection. Very gentle syringe aspiration prior to injection is also useful, although a negative result may be due to the collapse of the vessel wall against the needle orifice rather than the correct placement extravascularly. The use of short bevel needles permits better recognition of vascular entry.

A knowledge of the blood concentration profile for the agent and the technique employed will help to indicate the time when peak blood levels are likely to occur. Constant verbral contact and cardiorespiratory monitoring during this period is essential.

TREATMENT OF ACUTE LOCAL ANAESTHETIC TOXICITY

Airway

- Establish clear airway, suction if required

Breathing

- Oxygen with face mask
- Encourage adequate ventilation (prevent cycle of acidosis, increased uptake of local anaesthetic into CNS, and lowered seizure threshold)
- Artificial ventilation, if required

Circulation

- Elevate legs.
- Increase intravenous fluids if blood pressure falls.
- CVS support drugs, if decrease blood pressure & heart rate persists.

Drugs

- CNS depressant - Diazepam 5-10 mg

  Intravenous thiopentone 50mg, incremental doses until seizure ceases.
- Muscle relaxant

  Succinylcholine, 1 mg/kg – if inadequate control of ventilation with above measures (requires artificial ventilation and may necessitate intubation)

CVS support drugs

Atropine 0.6mg, intravenous if heart rate decreases.

Ephedrine, 12.5 –25 mg, intravenous, to restore adequate blood pressure.

Brachial plexus block is preferred for surgery over upper extremity as at this level instead of insertion of several needles a single thrust of a needle identifies not a particular nerve, but a particular fascial plane, within which the appropriate plexus lies. Infection into such a "interfascial compartment" of an appropriate volume of anaesthetic allows the solution, rather than the needle, "to seek out the nerve of the plexus".

Kulenkanff (1912) after experimentation on himself, devised supraclavicular approach to brachial block. The relative superficiality of plexus from this approach made patient particularly suitable. The supraclavicular block was first introduced in clinical practice by Labet (1928). Patrick (1940) starting upon a more accurate knowledge of surface anatomy of plexus, identified the proper land marks to minimize the ever present risk of pneumothorax. Macintosh and Mushin (1954) have modified the Patrick method by starting medially near the artery and then going laterally instead of starting laterally and working medially towards the artery. For the same reasons Ball (1962) has advised adopting a downward and inward direction.

Subsequently, there have been many modifications advocated. These modification vary mostly according to site, starting with the most proximal they are :-

1. Interscalene (Winnie 1970)
2. Parascalene (Vongvises & Panijayanond 1979)
3. Subclavian Perivascular (Winnie & Collins 1964)
4. Supraclavicular (Macintosh & Mushin 1967)
5. Infraclavicular (Raj et al 1973)
6. Axillary (De Jong 1961).

ANATOMY OF BRACHIAL PLEXUS

Brachial plexus supplies all of the motor and almost all of the sensory functions of the upper extremity. The plexus is formed from the anterior primary rami of the fifth, sixth, seventh and eighth cervical and the first thoracic nerves and frequently receives small contributing branches from the fourth (C_4) cervical and second thoracic (T_2) nerve.

After the nerve leave their respective intervertebral foramina they proceed anterolaterally and inferiorly to occupy the interval between the anterior and middle scalene muscles, where they unite to form three trunks-thus initiating the formation of brachial plexus. These trunks emerge from the interscalene space at the lower border of scaleneus muscles and continue anterolaterally and inferiorly to converge toward the upper surface of the first rib where they are closely grouped as superior, middle and inferior trunks, one above the other vertically. At the lateral edge.of the rib, each trunk divides into an anterior and posterior division which pass inferiorly to the mid portion of the clavicle to enter the axilla through its apex. These divisons reunite within the axilla to form three cords. All the posterior divisons of superior, middle and inferior trunk unite to form posterior cord, upper two anterior divison unite to form lateral cord and the lower anterior divison continues as medial cord. These are named according to their relationship with the second part of the axillary artery.

At the lateral border of the Pectoralis minor, the three cords break up to give rise to the peripheral nerves of the upper extremity. The lateral cord gives off the lateral head of median nerve and the musculocutaneous nerve, the medial cord gives off the medial head of median nerve, the ulnar nerve, the medial antebranchial, and the medial brachial cutaneous nerve and the posterior cord terminates as axillary and radial nerve.

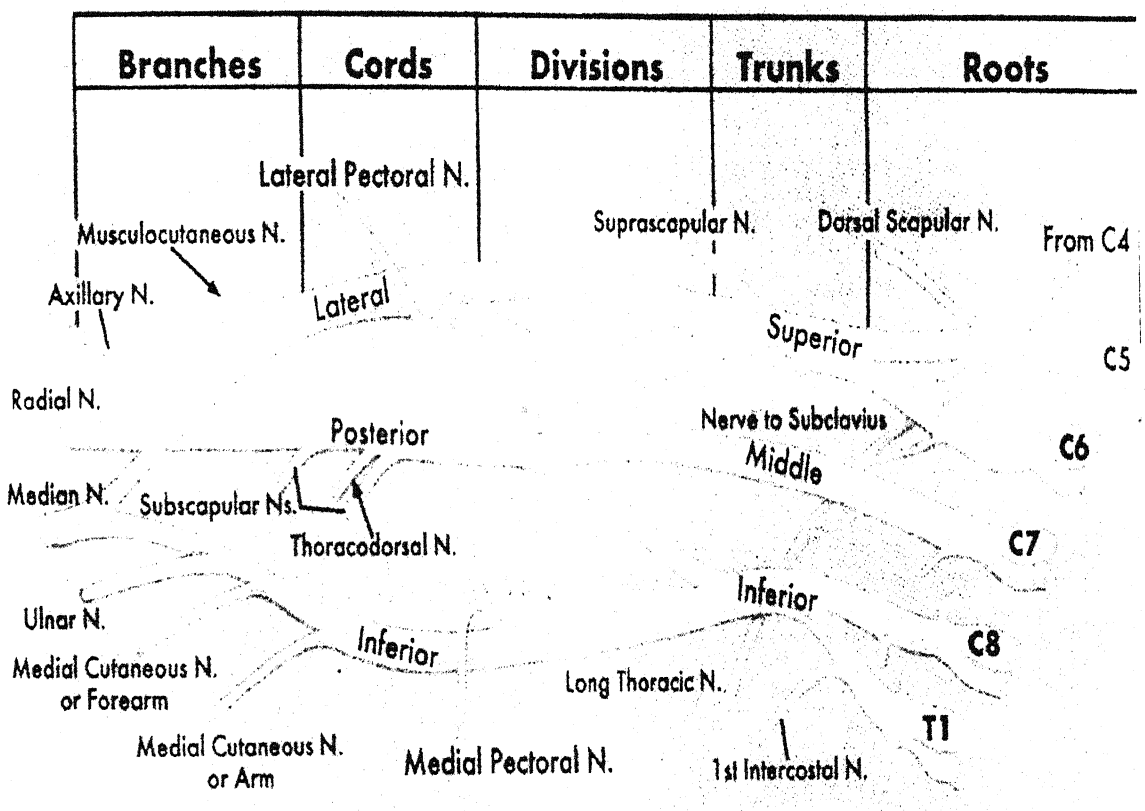


Fig 3 : Formation and Components of Brachial plexus

RELATIONS OF BRACHIAL PLEXUS

Anteriorly :- Skin, superficial fascia, platysma, supraclavicular branches of the cervical plexus, deep fascia, external juglar vein at the mid point of the clavicle and the clavicle in the front of its lower part. Scaleneus anterior in the front of its upper part.

Posteriorly :- Scaleneus medius and long thoracic nerve.

Inferiorly :- First rib and dome of the pleura

SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK:

It is the classical approach which relies on the predictable anatomy of the three major trunks of the plexus as they cross over the first rib between the insertion of the anterior and middle scaleneus just posterior to the subclavian artery.

Advantages

There are several advantages of supraclavicular block. It is blocked, where it is most compactly arranged at the level of three trunks. A small volume of solution is required and a quick onset is achieved. Also the technique can be performed with the arm being placed in any position. There is no danger of missing peripheral or proximal nerve branches because of failure of local anaesthetic spread.

Limitations and Problems

It has certain drawbacks. Considerable experience is required to perform brachial plexus block by this technique.

Contraindication

It is best avoided in the patients who are unco-operative, obese with unclear bony or muscular landmarks and those having respiratory problems.

Complications

(a) **Pneumothorax** – The most specific complication of the supraclavicular approach for blocking the brachial plexus block is pneumothorax. The frequency of occurrence is believed to be 0.5% to 6% and it decreases as the anaesthesiologist becomes more skilled. Bedder et al. (1988) reported an incidence of 1.6%. while N. Harley and J. Gjessing (1969) encountered complication rate of 5% of pneumothorax in their work. Tall and thin patients who characteristically have a high apical pleura usually account for the largest number of this complication.

The risk can be minimized by being careful, gentle, avoiding multiple indiscriminate probings and by the use of short and relatively fine needles. A pneumothorax must be suspected when there is dysponea, cough or pleuritic chest pain, but the diagnosis can only be confirmed by a chest radiograph.

(b) **Puncture of Subclavian Artery** – It is a serious complication due to the close proximity of blood vessels. N. Harley and J. Gjessing (1969) reported high incidence of arterial puncture in their cases. It can be best avoided by frequent aspiration, incremental injection and close observation. Symptoms and signs of toxicity appears very rapidly often before the injection has been completed.

Any complain about numbness of the tounge and circumoral tissues should warn the anaesthesiologist to stop the injection, otherwise it may lead to hypotension convulsions and even death. Undamaged arterial puncture with consequent haematoma formation remains another complication.

(c) **Block of the Phrenic nerve** – Occurs in 40% to 60% of the cases and usually causes no symptoms However, if a bilateral phrenic nerve block occurs in patient with underlying chest disease signs of hypoxia may result. Radiographic appearance of diaphgramatic paralysis has been reported in 36% of patients with interscalene and subclavian perivascular block. Fauar et al. (1961) reported incidence of 40% to 60% in their study while Matthes in 1969 found incidence of 23% with 2% mepivacaine hydrochloride in his work. On the contrary O. Schulte – Steinberg et al. (1970) encountered incidence of only 2% with 1.73% lignocaine carbonate solution.

(d) **Horner's Syndrome (Stellate Ganglion block)** It is seen in approximately 70% to 90% of the brachial plexus block when large volumes of the local anaesthetic solution (50 ml or more) are injected. The symptoms clears as the block dissipates. In the mean time patient should be assured.

(e) **Nerve damage or Neuritis** – It is rare but possible complication of all peripheral blocks including the supraclavicular technique for blocking the brachial plexus. The most frequent reason is the faulty positioning of the anaesthetized arm during surgery or in the immediate post-operative period. Trauma by the needle or prolonged ischaemia of the nerve due to vasoconstrictor drugs or too concentrated anaesthetic solution could also be a possible reason. These injuries may last for days to months. Physiotherapy and exercise are valuable to prevent muscle atrophy.

OBSERVATIONS

TABLE NO. I

Age and sex distribution of the cases.

Age (yrs)	No. of Patients									Total	% Age
	Group I (n=20)			Group II (n=20)			Group III (n=20)				
	Male	Female	Total	Male	Female	Total	Male	Female	Total		
20-30	9	1	10	8	1	9	9	2	11	**30**	50
31-40	4	1	5	3	1	4	2	2	4	**13**	21.66
41-50	2	1	3	3	0	3	2	0	2	**8**	13.33
51-60	2	0	2	4	0	4	3	0	3	**9**	15
Total	**17**	**3**	**20**	**18**	**2**	**20**	**16**	**4**	**20**	**60**	**100**

It is evident from the above table that maximum number of cases were from the age group 20-30 years (i.e. 50%). Out of 60 cases 51 cases (i.e. 85%) were male and 9 cases (i.e. 15%) were female.

TABLE NO. II

Distribution of the cases according to the surgical procedure

S.No.	Surgical procedure	Group I (n=20)	Group II (n=20)	Group III (n=20)	Total	%
1.	Radius plating	-	-	3	**3**	5
2.	Ulnar nailing	2	3	1	**6**	10
3.	Tension band wiring of olecranon	3	2	1	**6**	10
4.	Dynamic compression plate of humerus	7	5	8	**20**	33.33
5.	Radius plating & ulnar nailing	3	4	3	**10**	16.66
6.	Debridements	5	4	4	**13**	21.66
7.	Curettage & saucerization	-	2	-	**2**	3.33
Total		**20**	**20**	**20**	**60**	**100**

Table II indicates that cases were almost equally distributed among the groups and maximum number of cases underwent Dynamic compression plate for # humerus (i.e. 33.33%).

Percentage distribution of the cases according to the surgical procedure

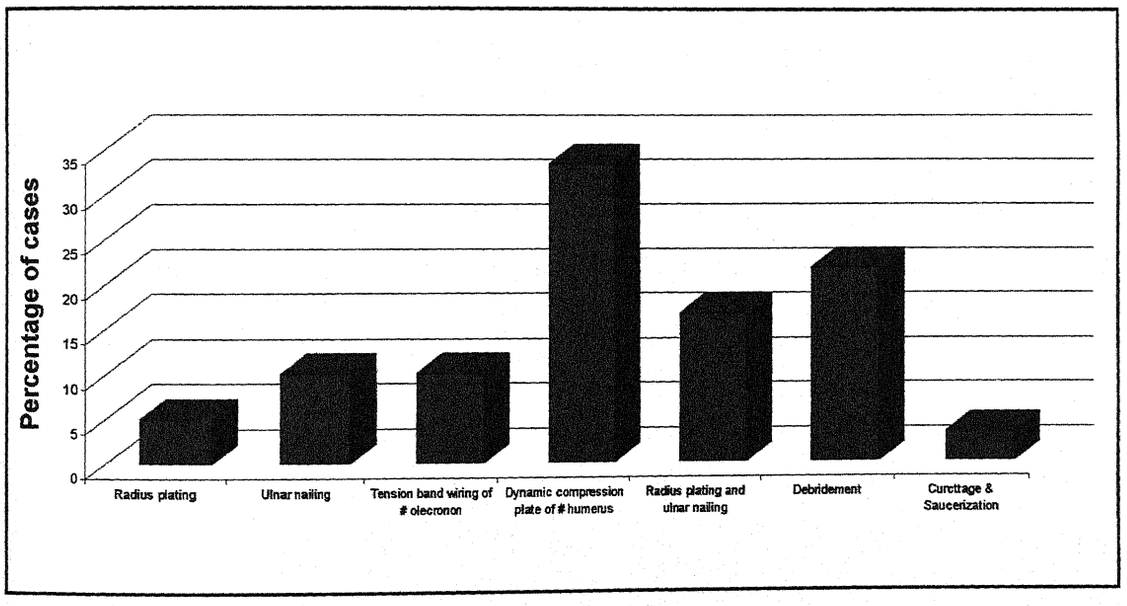


TABLE NO. III

Mean duration of the surgery in different study groups

(Minutes)

Group	Duration
Group I	71 ± 34.28
Group II	68.25 ± 29.39
Group III	70.75 ± 28.25

Table III shows that as per the duration of surgery was concerned, patients of all the three groups underwent almost similar duration of operative procedure.

Mean duration of the surgery in different study groups

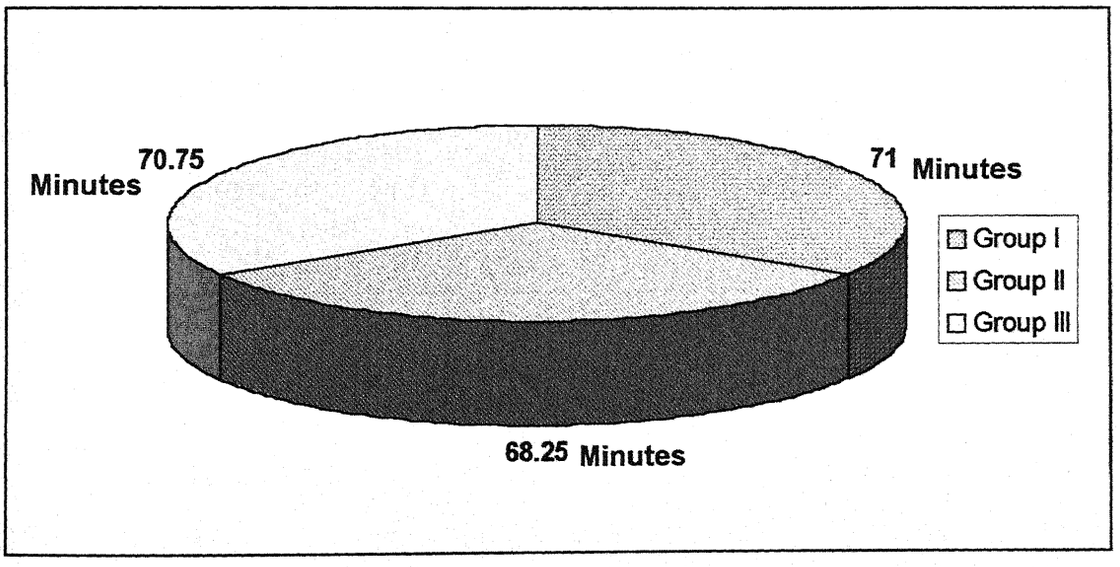


TABLE NO. IV

Changes in the mean pulse rate in different study groups

Statistical analysis of mean pulse rate (Beats/Min)

Group		Pre-Operative	15 minutes after injection	45 minutes after injection intra-operative	Post-operative
Group I (n=20)	**Mean ± S.D**	87.9 ± 6.20	86.7^{ns} ± 6.39	84.6^{***} ± 3.88	86.8^{ns} ± 6.56
Group II (n=20)	**Mean ± S.D**	91.8 ± 8.65	86.5^{***} ± 6.79	82.7^{***} ± 8.23	88.7^{***} ± 7.32
Group III (n=20)	**Mean ± S.D**	91.5 ± 5.61	87.2^{**} ± 4.80	85.5^{***} ± 7.56	89.4^{*} ± 4.05

$p > .05^{ns}$, $p < .05^{*}$, $p < .01^{**}$, $p < .001^{***}$

Table No. IV indicates that the pre-operative pulse rate among the groups were similar and within normal range. After 15 minutes of injection of local anaesthetic drug, highly significant fall was seen in group II with only significant and no change observed in group III and group I respectively. Maximum fall in the pulse rate was seen 45 minutes after the performance of block in all the groups. Later on in post operative period the mean pulse rate in all the three groups rise from their intra-operative mean values but was still below their preoperative mean pulse rate values.

Changes in the mean pulse rate in different study groups

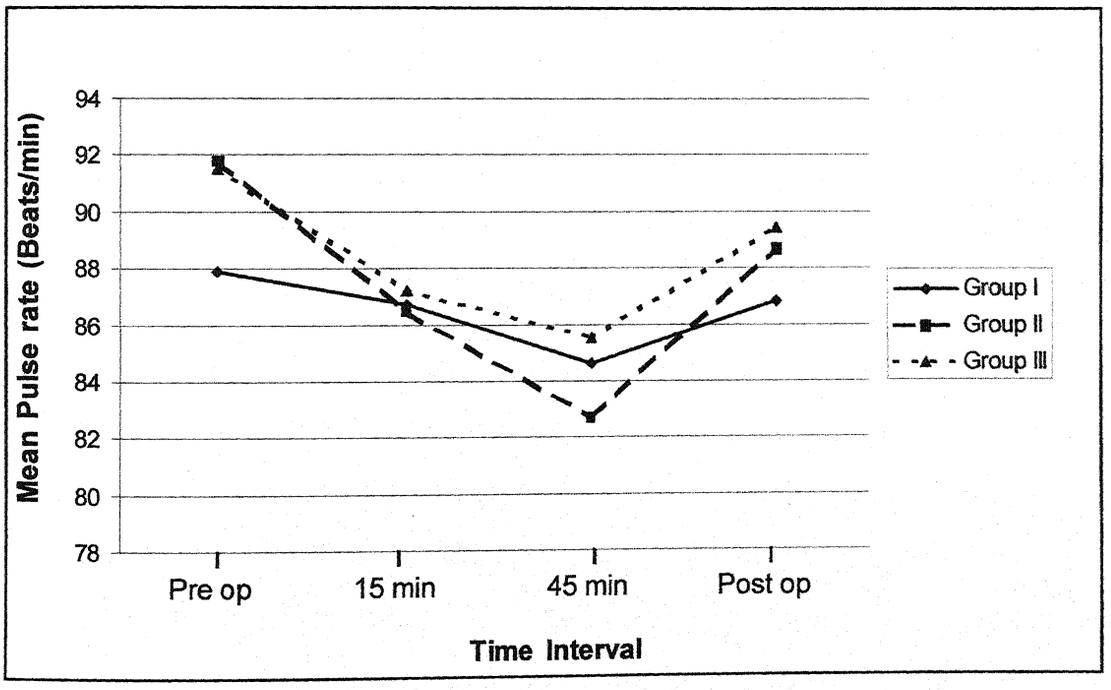


TABLE NO. V

Changes in the mean respiratory rate in different study groups

Statistical analysis of mean respiratory rate (Breaths/Min)

Group		Pre-Operative	15 minutes after injection	45 minutes after injection intra-operative	Post-operative
Group I (n=20)	**Mean ± S.D**	19.8 ± 1.43	19.4^{ns} ± 1.46	18.7^{**} ± 1.34	19.5^{ns} ± 1.57
Group II (n=20)	**Mean ± S.D**	20.6 ± 1.7	19.2^{***} ± 1.46	19.0^{***} ± 1.5	19.2^{**} ± 2.06
Group III (n=20)	**Mean ± S.D**	20.6 ± 1.14	20.0^{ns} ± 1.17	19.2^{***} ± 1.19	20.3^{ns} ± 0.97

$p > .05^{ns}$, $p < .05^{*}$, $p < .01^{**}$, $p < .001^{***}$

Above table No. V shows that a highly significant fall in the respiratory rate from their preoperative value was observed in group II after 15 minutes of injection of local anaesthetic agent. While group I and III cases observed insignificant change in their mean respiratory rate value. Group I and group III registered significant and highly significant fall in the respiratory rate 45 minutes after injection intraoperatively. Group II continued with the highly significant fall in the mean respiratory rate value at 45 minutes of injection of the drug In post operative period significant fall in the respiratory rate from their preoperative value was noticed in group II cases only while group I and group III cases had insignificant changes.

Changes in the mean respiratory rate in different study groups

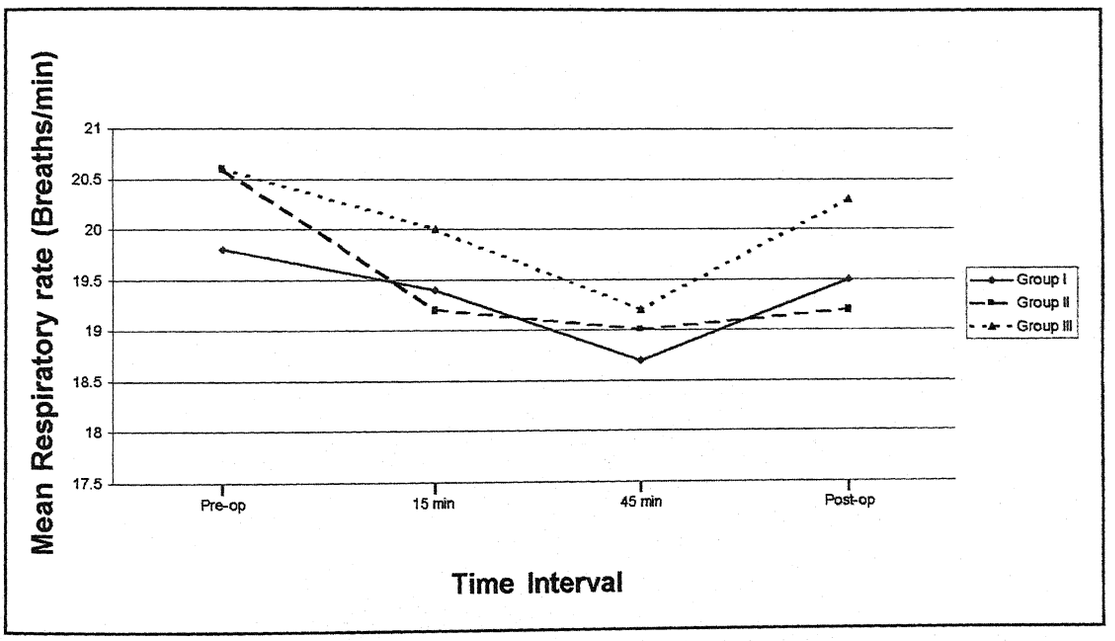


TABLE NO. VI

Changes in the mean arterial pressure in different study groups.

Statistical analysis of mean arterial pressure (mm Hg)

Group		Pre-Operative	15 minutes after injection	45 minutes after injection intra-operative	Post-operative
Group I (n=20)	**Mean ± S.D**	96.62 ± 5.92	96.41^{ns} ± 5.59	95.99^{ns} ± 6.79	96.42^{ns} ± 5.94
Group II (n=20)	**Mean ± S.D**	95.13 ± 5.65	94.26^{ns} ± 6.04	93.83^{ns} ± 5.88	94.39^{ns} ± 5.5506
Group III (n=20)	**Mean ± S.D**	93.76 ± 5.53	93.09^{ns} ± 4.93	93.83^{ns} ± 4.89	93.76^{ns} ± 5.51

$P>.05^{ns}$

Table No. VI shows no significant change in the mean arterial pressure of the groups from their preoperative values at 15,45 minutes interval as well as post operatively.

Changes in the mean arterial pressure in different study groups.

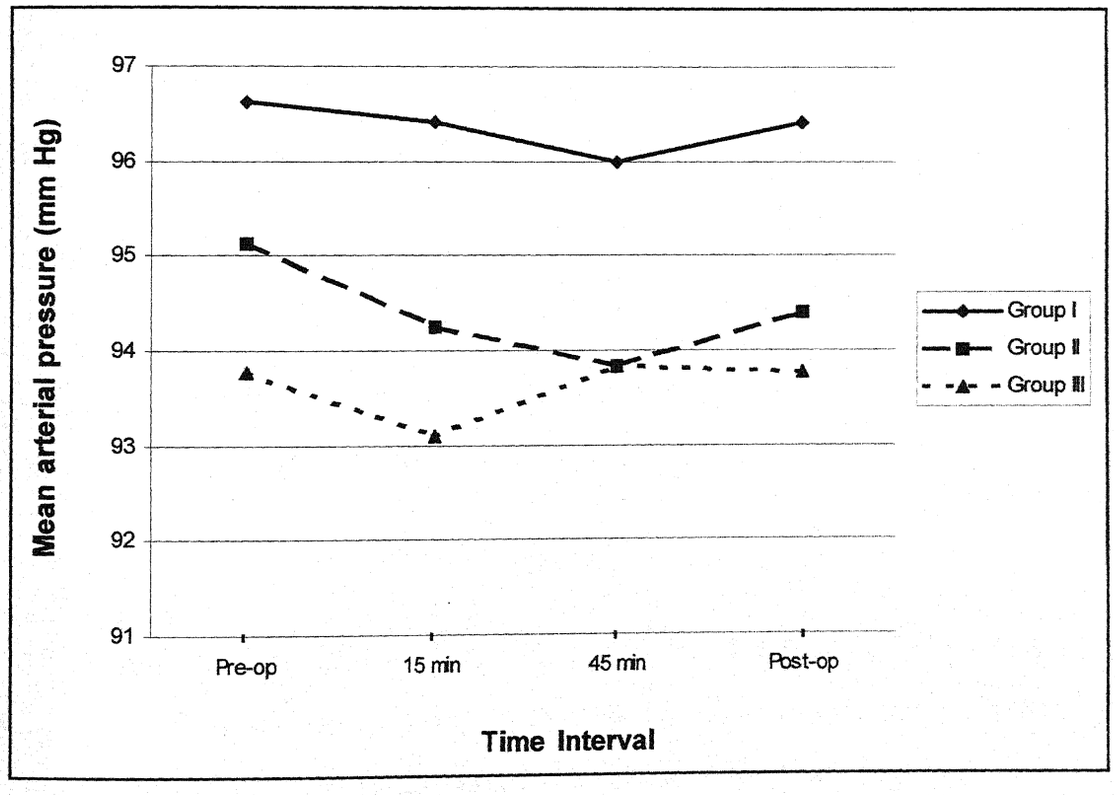


TABLE NO. VII

Changes in the mean arterial oxygen saturation in the different study groups. Statistical analysis of mean arterial oxygen saturation (%)

Group		Pre-Operative	15 minutes after injection	45 minutes after injection intra-operative	Post-operative
Group I (n=20)	**Mean ± S.D**	97.65 ± 0.58	97.8^{ns} ± 0.40	97.55^{ns} ± 0.50	97.65^{ns} ± 0.48
Group II (n=20)	**Mean ± S.D**	97.45 ± 0.60	97.65^{ns} ± 0.24	97.6^{ns} ± 0.50	97.6^{ns} ± 0.50
Group III (n=20)	**Mean ± S.D**	97.2 ± 0.69	97.6^{ns} ± 0.50	97.7^{ns} ± 0.47	97.6^{ns} ± 0.50

$P>.05^{ns}$

Table No. VII shows statistical analysis of mean arterial oxygen saturation of all the three groups. After analysis values of mean arterial oxygen saturation were found to be insignificant from their preoperative values in all the three groups at various intervals.

Changes in the mean arterial oxygen saturation in the different study groups

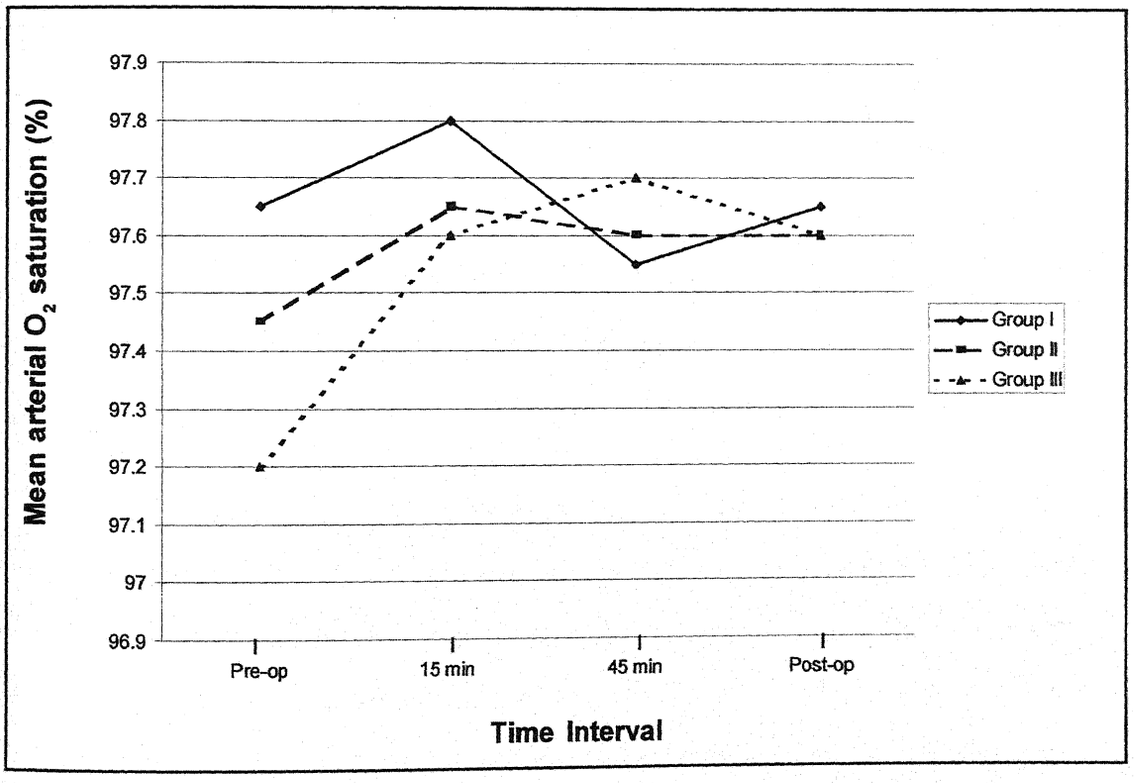


TABLE NO. VIII

Latency of Sensory block

Group I Vs Group II

(Minutes)

Parameter	Group I (n=20)	Group II (n=20)
Temperature (Mean ± S.D)	5.8 ± 2.44	3.25 ± 1.48***
Touch (Mean ± S.D)	9.1 ± 3.64	5.1 ± 1.83***
Pin prick (Mean ± S.D)	12.1 ± 4.43	6.7 ± 2.07***
Pressure (Mean ± S.D)	18.35 ± 6.80	10.35 ± 2.97***

P< .001***

Table no. VIII shows comparison of latency of sensory block between group I and group II. Duration for the latency of sensory block was calculated from the time of injection to no response to temperature, touch, pin prick and pressure sensations. Above table shows mean duration of loss of temperature, touch, pinprick and pressure sensation in group I and group II. When both the groups were compared statistically, taking in consideration all the parameters separately, the probability of the values were found to be highly significant.

The mean duration of complete onset of sensory block in group I was 18.35 ± 6.80 minutes and in group II 10.35 ± 2.97 minutes.

Latency of Sensory block
Group I Vs Group II

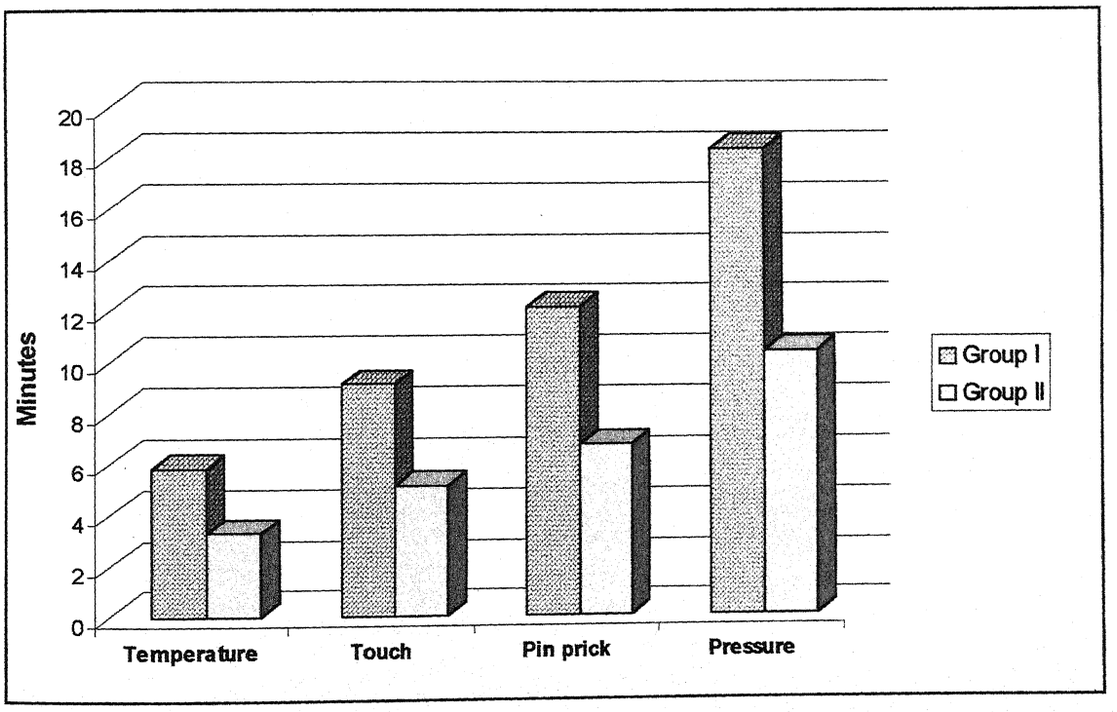


TABLE NO. IX

Latency of Sensory block

Group I Vs Group III

(Minutes)

Parameter	Group I (n=20)	Group III (n=20)
Temperature (Mean ± S.D)	5.8 ± 2.44	3.8 ± 1.43**
Touch (Mean ± S.D)	9.1 ± 3.64	5.9 ± 2.46**
Pin prick (Mean ± S.D)	12.1 ± 4.43	8.1 ± 3.68**
Pressure (Mean ± S.D)	18.35 ± 6.80	12.85 ± 3.91**

P< .01**

Table no. IX shows comparison of latency of sensory block between group I & group III. Group III when was compared with group I the probability of the values for all the parameters regarding latency of sensory block was found to be more significant. Mean duration for the achievement of complete sensory block in group III was 12.85 ± 3.91 minutes.

Latency of Sensory block
Group I Vs Group III

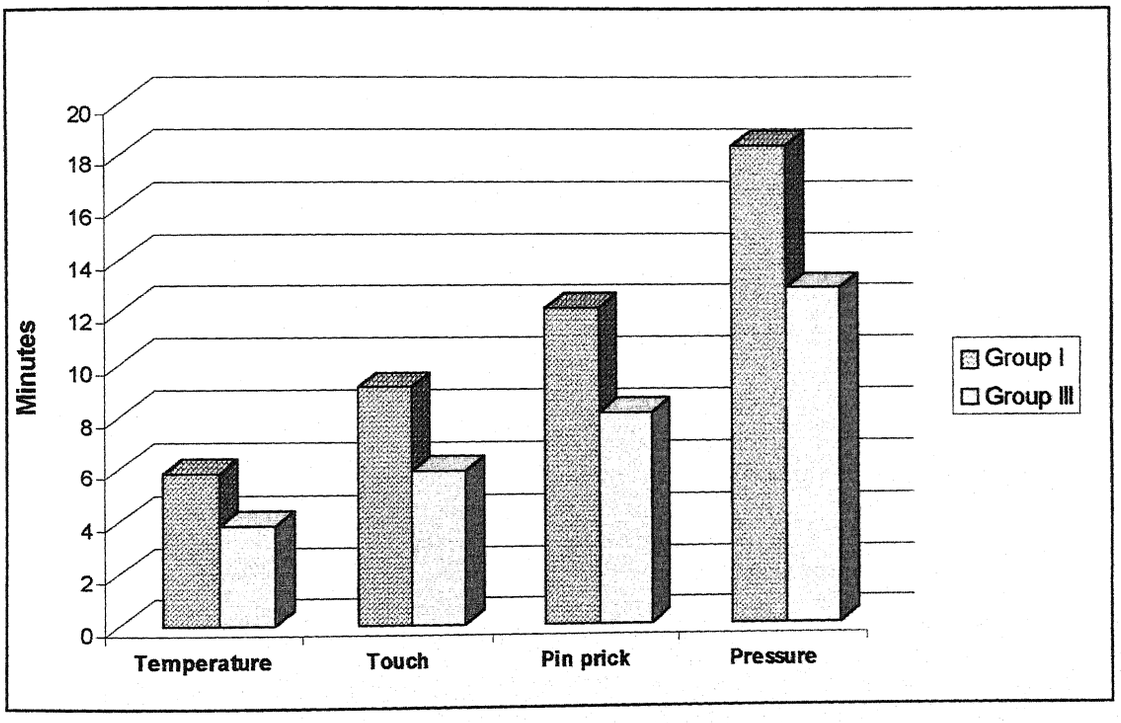


TABLE NO. X

Latency of Sensory block

Group II Vs Group III
(Minutes)

Parameter	Group II (n=20)	Group III (n=20)
Temperature (Mean ± S.D)	3.25 ± 1.48	3.8 ± 1.43 ^{ns}
Touch (Mean ± S.D)	5.1 ± 1.83	5.9 ± 2.46 ^{ns}
Pin prick (Mean ± S.D)	6.7 ± 2.07	8.1 ± 3.68 ^{ns}
Pressure (Mean ± S.D)	10.35 ± 2.97	12.85 ± 3.91 ^{ns}

$P > .05^{ns}$

Table no. X shows comparison of latency of sensory block between group II & group III When both group II and group III were compared taking in consideration all the parameters separately, the values were found to be insignificant.

Latency of Sensory block
Group II Vs Group III

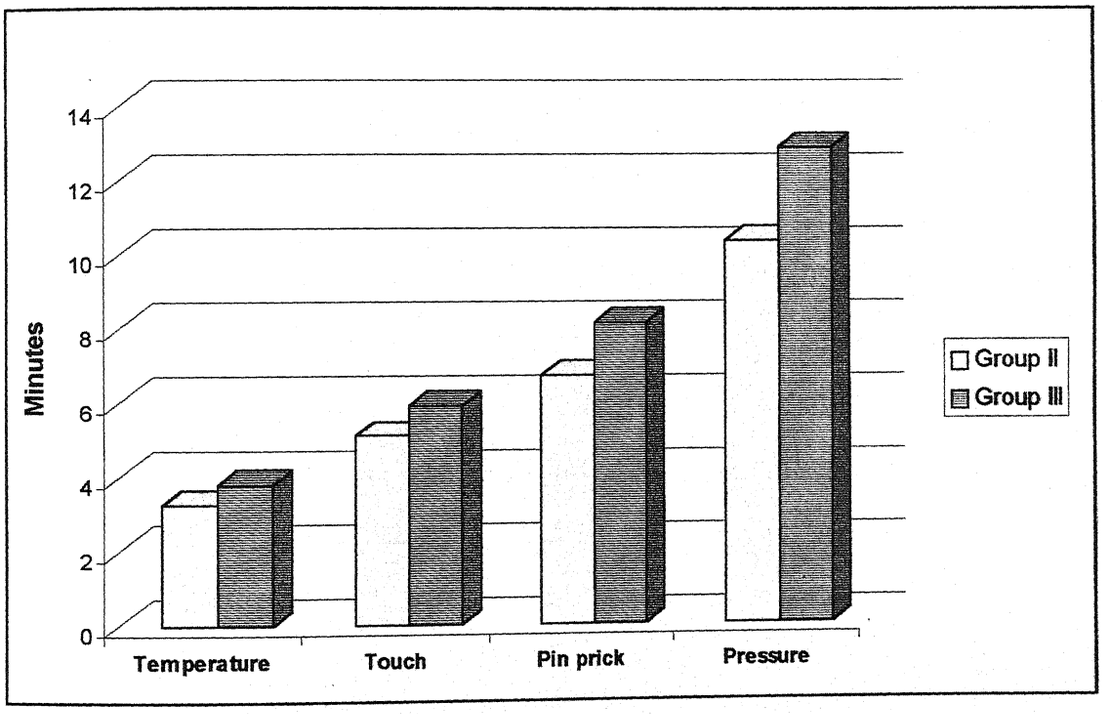


TABLE NO. XI

Latency of Motor block
Group I Vs Group II
(Minutes)

Parameter	Group I (n = 20)	Group II (n = 20)
Paresis (Mean ± S.D)	20.65 ± 5.22	12.2 ± 2.44***
Paralysis (Mean ± S.D)	30.15 ± 8.56	22.55 ± 8.58*

$P < .05^{*}$, $P<.001^{***}$

Table no. XI shows comparison of latency of motor block between group I & group II. In the assessment of latency of motor block the duration for the onset of paresis was taken from the time of injection to the loss of dorsiflexion of wrist joint and paralysis from the time of injection to the complete loss of finger movements..

The mean duration of paresis by group II was found to be 12.2 ± 2.44 minutes. When this value was compared with mean duration of the attainment of paresis for group I which is 20.65 ± 5.22 minutes, the value was found to be highly significant.

Complete paralysis was achieved in a mean duration of 22.55 ± 8.58 minutes by group II and the value was found to be only significant in relation to group I who attained complete paralysis in a mean duration of 30.15 ± 8.56 minutes.

Latency of Motor block
Group I Vs Group II

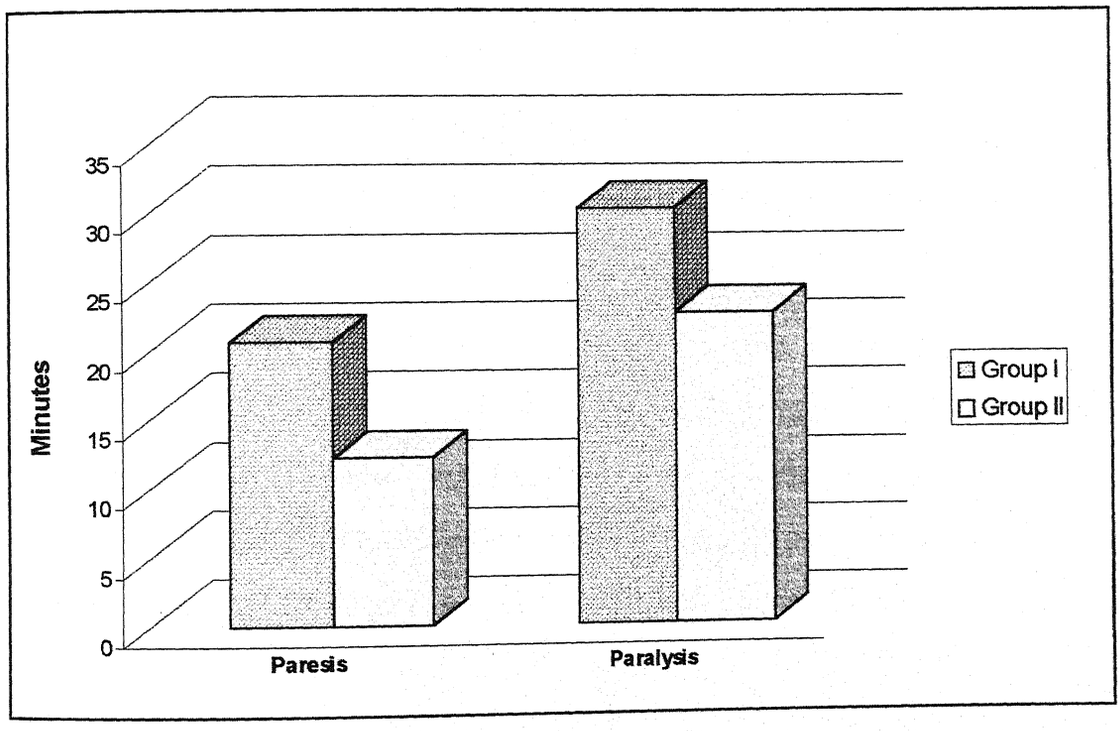


TABLE NO. XII

Latency of Motor block

Group I Vs Group III
(Minutes)

Parameter	Group I (n = 20)	Group III (n = 20)
Paresis (Mean ± S.D)	20.65 ± 5.22	13.8 ± 4.74***
Paralysis (Mean ± S.D)	30.15 ± 8.56	23.3 ± 7.09*

P < .05*, P<.001***

As shown in the above table no XII, the mean duration of achievement of paresis for group III is 13.8 ± 4.74 minutes which is highly significant when compared with the value of mean duration of achievement of paresis for group I. On the other hand when the value for mean duration of onset of complete paralysis for group III was compared with group I it was found to be only significant.

Latency of Motor block
Group I Vs Group III

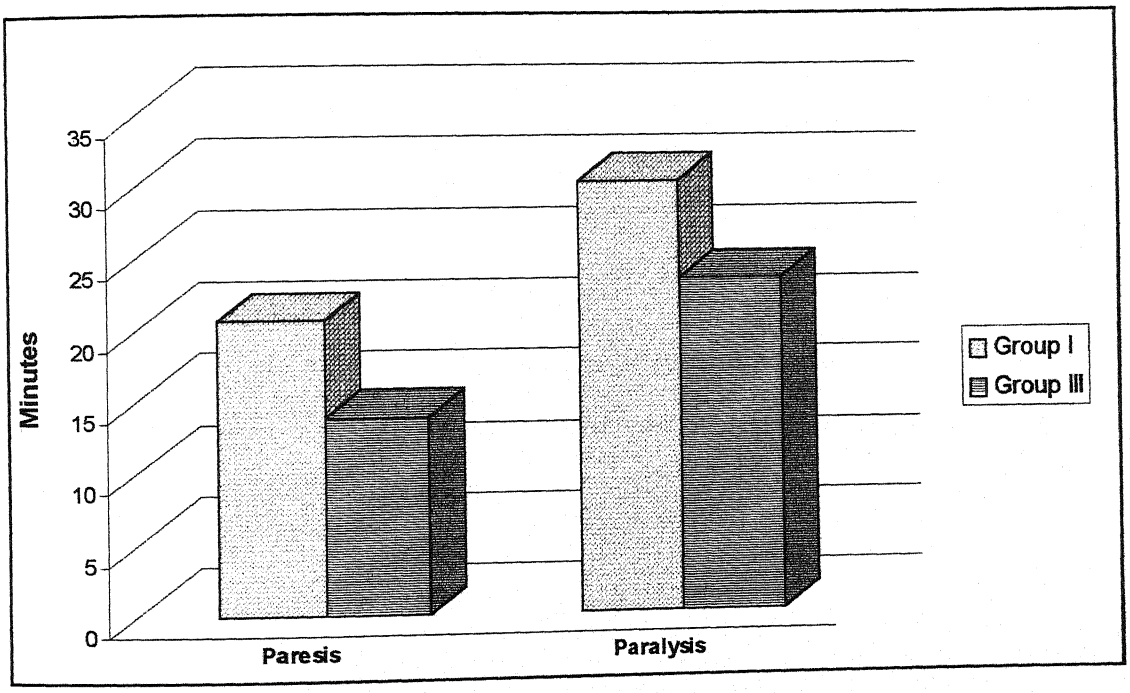


TABLE NO. XIII

Latency of Motor block
Group II Vs Group III
(Minutes)

Parameter	Group II (n = 20)	Group III (n = 20)
Paresis (Mean ± S.D)	12.2 ± 2.44	13.8 ± 4.74^{ns}
Paralysis (Mean ± S.D)	22.55 ± 8.58	23.3 ± 7.09^{ns}

$P > .05^{ns}$

Table No XIII shows mean duration of attainment of both paresis and paralysis by group II and III, when their values were statistically compared, they were found to be insignificant.

Latency of Motor block
Group II Vs Group III

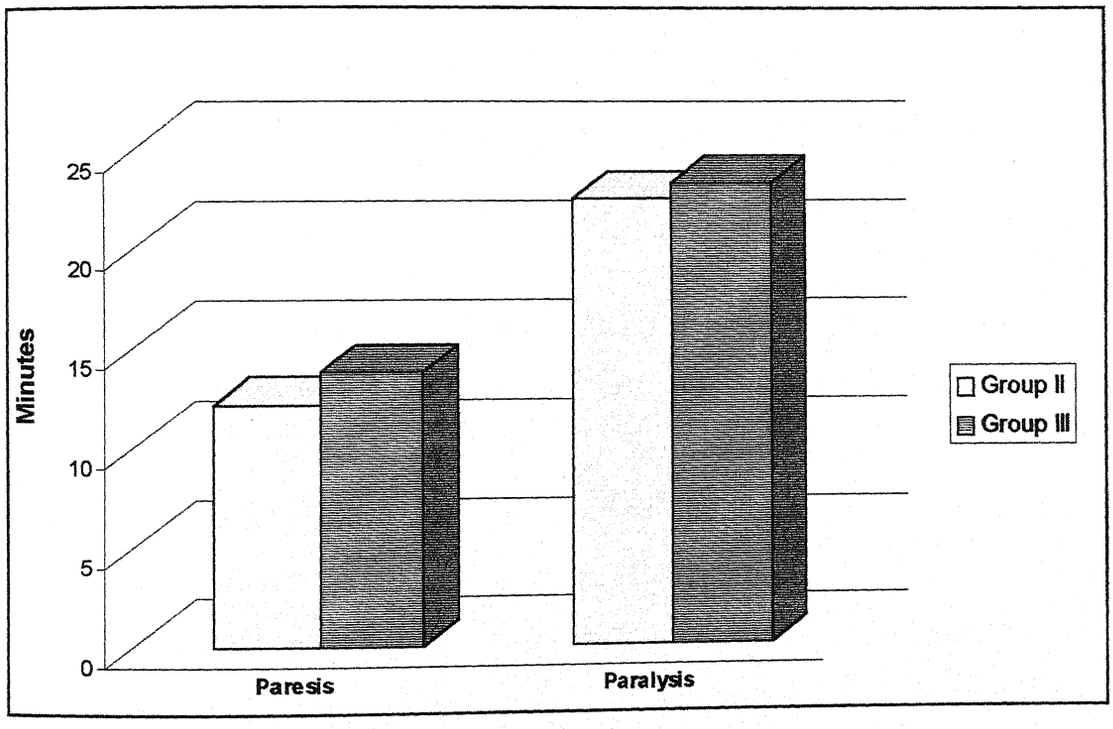


TABLE NO. XIV

Duration of block

Group I Vs Group II
(Minutes)

Type of Block	Group I (n = 20)	Group II (n = 20)
Sensory (Mean ± S.D)	87.1 ± 17.10	101.95 ± 11.82 ^{ns}
Motor (Mean ± S.D)	107.4 ± 13.31	117.55 ± 9.85 **

$P > .05^{ns}$, $P<.01^{**}$

Table no XIV shows comparison of duration of block between group I and group II. Duration of sensory block was taken from the time of complete onset of sensory block till the patient respond to pin prick and the duration of motor block was taken from the time of loss of finger movements till the patient started moving his fingers.

P>.05 in the above table shows that values for the mean duration of sensory block of both the groups I & II when compared were found to be insignificant, while values for the mean duration of motor block were more significant P <.01.

Duration of block
Group I Vs Group II

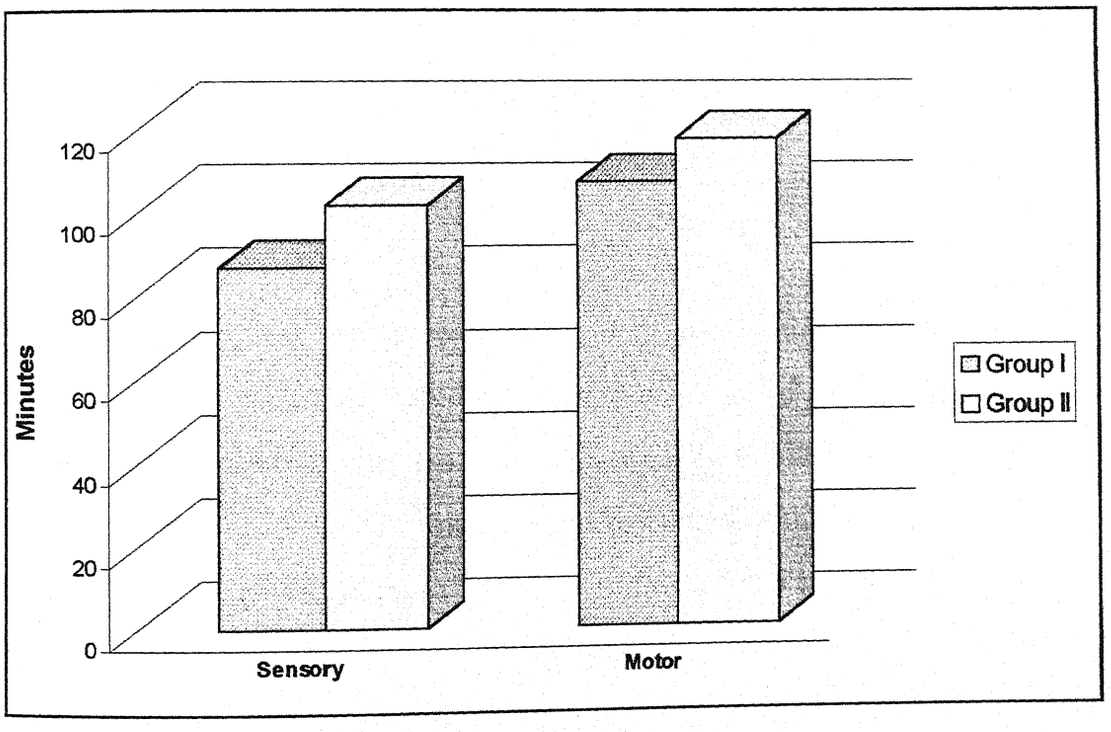


TABLE NO. XV

Duration of block
Group I Vs Group III
(Minutes)

Type of Block	Group I (n = 20)	Group III (n = 20)
Sensory (Mean ± S.D.)	87.1 ± 17.10	100.7 ± 12.84 ^{ns}
Motor (Mean ± S.D.)	107.4 ± 13.31	115.9 ± 9.49*

$P > .05^{ns}$, $P<.05^{*}$

Table No. XV shows comparison of mean duration of sensory and motor block in group III with group I. Comparison of mean duration of sensory block between the two groups showed probability of >.05. Mean duration of motor block for group I when compared with mean duration of motor block for group III the difference was found to be significant p<.05.

Duration of block
Group I Vs Group III

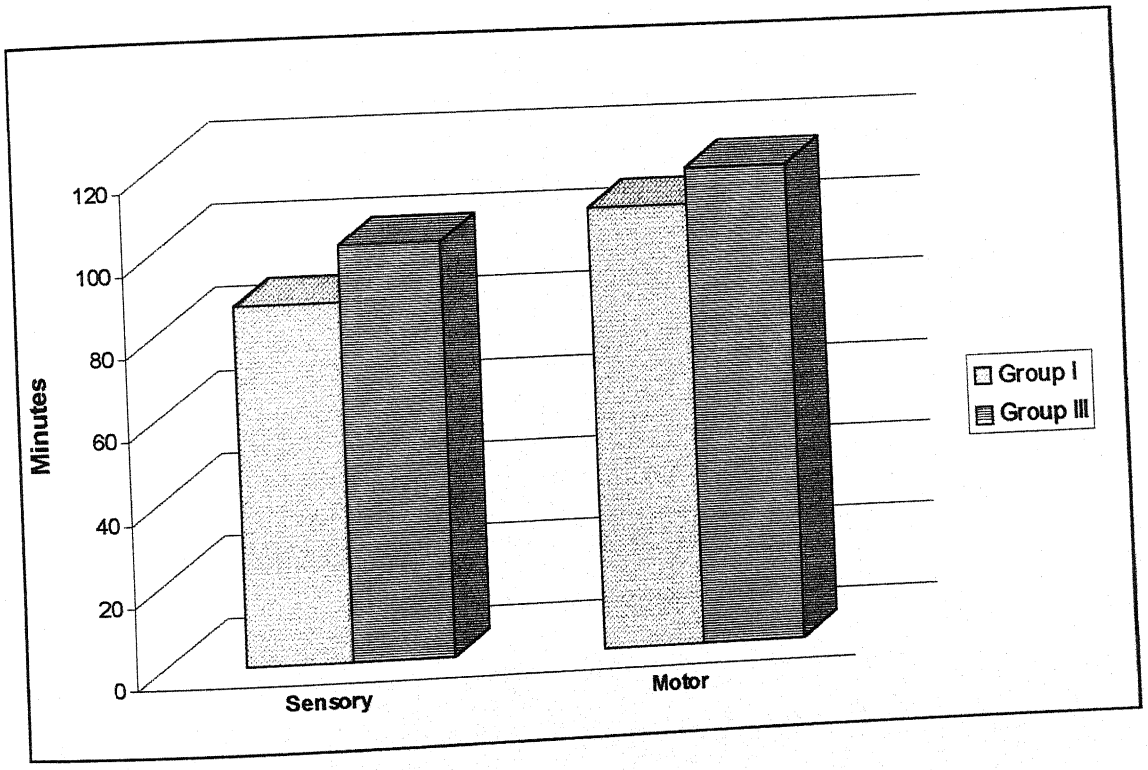


TABLE NO. XVI

Duration of block

Group II Vs Group III
(Minutes)

Type of Block	Group II (n = 20)	Group III (n = 20)
Sensory (Mean ± S.D.)	101.95 ± 11.82	100.7 ± 12.84 ^{ns}
Motor (Mean ± S.D.)	117.55 ± 9.85	115.9 ± 9.49 ^{ns}

$P > .05^{ns}$

Table no. XVI shows comparison of duration of block between group II and group III. In group II mean value of duration of sensory block was found to be 101.95 ± 11.82 minutes and motor block 117.55 ± 9.85 minutes while in group III mean value of duration of sensory block was 100.7 ± 12.84 minutes and motor 115.9 ± 9.49 minutes. P >.05 shows that the difference in duration of both sensory and motor block between these two groups was statistically insignificant.

Duration of block
Group II Vs Group III

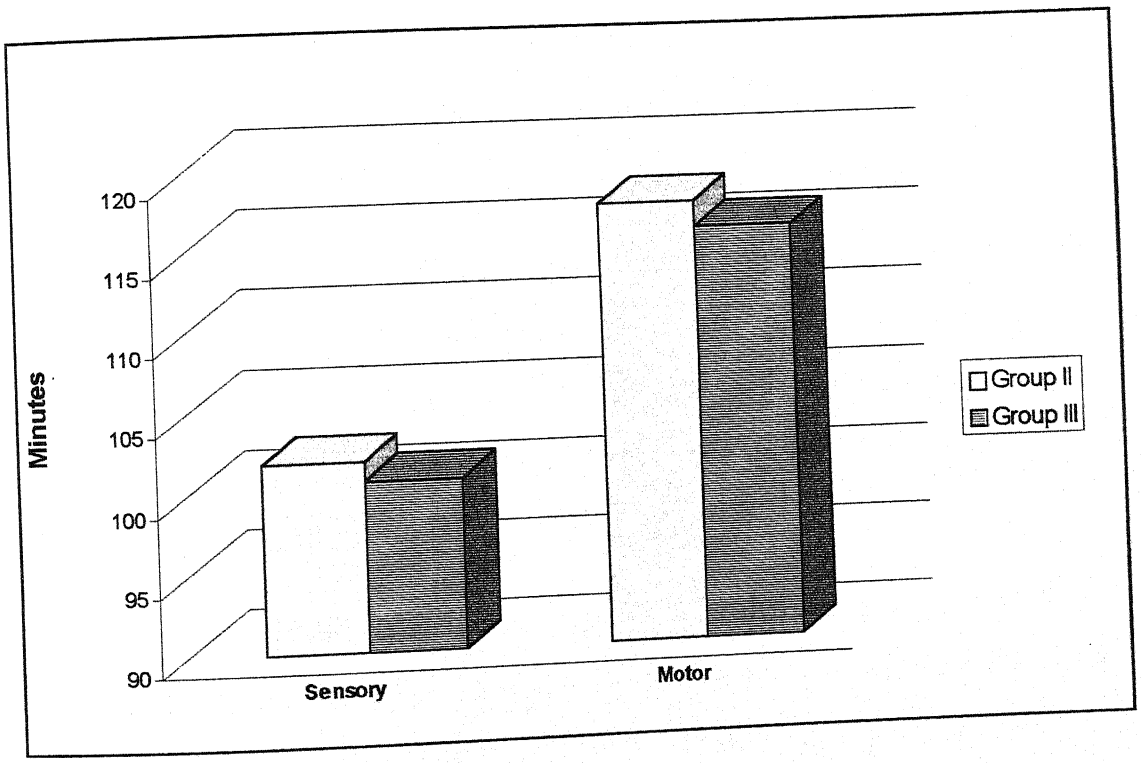


TABLE NO. XVII

Quality of block

Group	Complete	%	Incomplete	%	Failed	%
Group I (n = 20)	7	35	13	65	0	-
Group II (n = 20)	16	80	4	20	0	-
Group III (n = 20)	15	75	5	25	0	

Quality of the block was graded into three. Complete, Incomplete and Failed. Table no XVII shows % of quality of block in the different groups.

Block was said to be complete when all the parameters of both sensory and motor block was achieved within thirty five minutes of duration from the time of injection. Out of 20 cases in each group . 7 cases (ie 35%) had complete block in group I, 16 cases (ie 80%) in group II and 15 cases (ie. 75%) in group III.

Incomplete block was the one when any of the parameters other than pin prick could not be achieved within thirty five minutes of duration from the time of injection. Maximum number of incomplete block that is 13 out of 20 cases (ie 65%) was seen in group I, next in group III 5 out of 20 cases (ie 25%) and lastly in group II that is 4 out of 20 cases (i.e 20%).

Block was declared failed when none of either motor or sensory block parameters including pin prick could be achieved in duration more than thirty five minutes. Failed block was not seen in either of the groups.

Quality of block

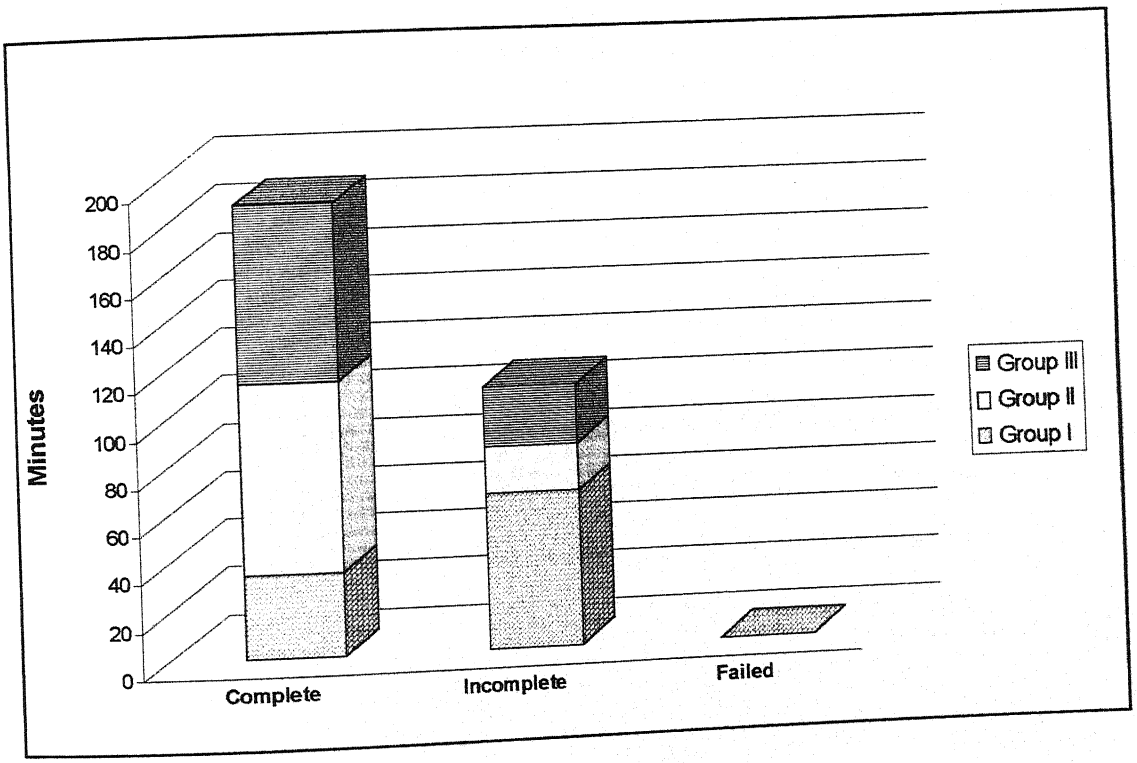


TABLE NO. XVIII

Complications

Type of Complication	Group I (n = 20)	Group II (n = 20)	Group III (n = 20)
Arterial Puncture	2	2	1
Pneumothorax	-	-	-
Phrenic. N. Palsy	-	-	-
Toxicity	-	-	-

Arterial puncture which is a common complication was encountered twice in both group I and II and only once in group III. Pneumothorax, phrenic N. palsy and toxicity was not observed in any of the cases of either groups.

DISCUSSION

DISCUSSION

The present study is an attempt to compare relative efficacy of lignocaine hydrochloride at different pH with respect to brachial plexus block. The study was carried out on sixty healthy adult cases of A.S.A. grade I & II status, between 20-60 years age group scheduled for upper extremity surgery. Patients were randomly allocated into three groups of twenty cases each. pH of the groups were constituted by adding required amount of sodium-bi-carbonate solution. pH of group I was 3.21, group II-6.21 and group III-6.67.

The demographic and operative data in all the groups were comparable. Maximum number of the patients in the groups were from 20-30 years age group. Group I 50%, group II 45% and group III 55%. Male/Female ratio in the study was 51:9 with 17:3 in group I, 18:2 in group II and 16:4 in group III. Distribution of the cases among the groups was also almost identical with 33.33% of the cases in the study underwent Dynamic compression plate for # humerus. As per the duration of surgery was concerned the operative procedures in all the groups was of almost similar duration. Mean duration of surgery in group I was 71 $\pm$ 34.28 minutes, in group II 68.25 $\pm$ 29.39 minutes and in group III 70.75 $\pm$ 28.25 minutes.

Latency of Sensory Block

Temperature, touch, pinprick and pressure sensation was taken as the parameters for estimating the latency of sensory block in our study. It was calculated from the time of injection to the complete loss of all the above sensory parameter. We found that in all groups the first sensory parameter to be lost was temperature which was followed by touch, pinprick and

lastly pressure sensation. Cases in group II took minimum time in comparison to cases in group I and group III for the loss of all the sensory parameters indivually.

In group I cases, the temperature sensation was lost in 5.8 $\pm$ 2.44 minutes, touch in 9.1 $\pm$ 3.64 minutes, pinprick in 12.1 $\pm$ 4.43 minutes and pressure sensation in 18.35 $\pm$ 6.80 minutes from the time of injection of the drug while in group II the temperature was lost in 3.25 $\pm$ 1.48 minutes, touch in 5.1 $\pm$ 1.83 minutes, pinprick in 6.7 $\pm$ 2.07 minutes lastly pressure in 10.35 $\pm$ 2.97 minutes from the time of injection. Group III recorded loss of temperature sensation in 3.8 $\pm$ 1.43 minutes, touch in 5.9 $\pm$ 2.46 minutes, pinprick in 8.1 $\pm$ 3.68 minutes and pressure in 12.85 $\pm$ 3.91 minutes. In group I complete sensory block was obtained in the mean duration of 18.35 $\pm$ 6.80 minutes, in group II in 10.35 $\pm$ 2.97 minutes and in group III in 12.85 $\pm$ 3.91 minutes from the time of performance of the block. In group II decrease in the latency of sensory block was by 43.59% while in group III this decrease was only by 29.97% from the control value. Mean values of the different parameters for the latency of sensory block was found to be highly significant ($P<.001$) for group II and more significant ($P<.01$) for group III in comparison to group I. Mean values of different parameters of latency of sensory block in group II and group III when compared with each other were found to be insignificant, confirming that further alkalinization of local anaesthetic drug used in group III cases resulted no further advantage.

Present study co-relates well with the findings of earlier studies done with lignocaine hydrochloride solution at different pH on brachial plexus block. The 43.59% reduction in latency of sensory block observed in our study is almost similar to 45% reduction noted in the study done by

Radha Sukhani and Alon P. Winnie (1987). In their study they compared 1% Lidocaine hydrochloride with 1.1% lidocaine carbonate. The reduction in the onset time they attributed to be due to greater rapidity of spread. tissue penetration and intraneural diffusion more with the carbonate than with the hydrochloride salt.

O. Schulte-Steinberg, J Hartmuth, L Schult (1970) when in their study compared 1.73% carbonated lignocaine (pH 6.5) with 2% lignocaine hydrochloride solution, (p^H 4) they found latency for complete analgesia to be averaged 4-5 minutes for carbonated solutions and 4-22 minutes for hydrochloride solutions. Despite various technical difficulties surgery was started within 10 minutes of first injection with carbonated solution while with hydrochloride solution a waiting period of approximately 15 minutes was required.

Bromage P. R (1971) noticed 42% reduction in the latency of anaesthesia using 2.2% lignocaine carbonate solution.

Mc. Clure and Scotl (1981) demonstrated a similar reduction in latency and increase in spread of anaesthesia produced by the carbonate salt of bupivacaine compared to the hydrochloride salt.

AV Gormley WP, Hill DA, Murray JM, Fee JP (1996) demonstrated in their study using 1.5% lignocaine with adrenaline (pH 4.2) and alkalinized 1.5% lignocaine with adrenaline (pH 7.2) on axillary plexus block that there was a significant reduction in the time to useful anaesthesia and reduced requirement for the adjuvants in the alkalinized group.

Latency of motor block

In assessing the latency of motor block the onset time of paresis was the time from injection to loss of dorsiflexion of wrist joint and paralysis from the time of injection to complete loss of finger movements.

Mean value for the onset time of paresis is 20.65 $\pm$ 5.22 minutes in group I, 12.2 $\pm$ 2.44 minutes in group II and 13.8 $\pm$ 4.74 minutes in group III. Cases in group II and cases in group III showed reduction in the onset time of paresis by 40.92% and 33.17% respectively from the control group. This reduction in the onset time for paresis was statistically highly significant ($P < .001$).

Complete paralysis was achieved in a mean duration of 30.15 $\pm$ 8.56 minutes in group I, 22.55 $\pm$ 8.58 minutes in group II and 23.3 $\pm$ 7.09 minutes in group III. Mean values of the onset time of complete paralysis in group II and in group III were found to be only significant ($P < .05$) with reduction in the onset time by 25.20% and 22.71% respectively form the control values.

Group II when compared with Group III in reference to the mean values for the onset time of paresis and paralysis, the values were insignificant. ($P > .05$). Finally the minimum latency of motor block in our study was seen in group II with onset of paresis being significantly faster than the onset of paralysis.

Radha Sukhani et al. (1987) in their study observed that the onset of paralysis is significantly faster with the carbonated lidocaine (pH. 6.8) than with the hydrochloride (p^H 6.5). They found a 30% reduction in onset time for paresis and 50% reduction in onset time for paralysis with carbonated lignocaine.

In our study the time for paresis was almost equal to the time for onset of sensory block in all the cases in each group. This was perhaps because of the fact that as local anaesthetic solution penetrates the epineural barrier, the first fibres encountered are motor and so blocking processes begins in motor fibres before the local anaesthetic has even reached any sensory fibre.

R Martin I, Beauregard et al. (1987) while comparing lidocaine hydrochloride, lidocaine hydrocarbonate and mepivacaine in brachial plexus block found that there was no significant difference in onset of block between lidocaine carbonate and hydrochloride groups but faster onset.

Quinlan et at. (1992) while working with alkalinized mepivacaine on axillary block concluded that no significant difference in time to onset of paresis was noted but it significantly shortned the time to onset of both proximal and distal paralysis.

Chow et al. (1998) showed in their study that alkalinized lidocaine (p^H 7.15) did not improve the onset of motor block after axillary brachial plexus anaesthesia.

Quality of Block

Quality of the block was graded into three. Complete, Incomplete and Failed.

Block was said to be complete when all the parameters of both sensory and motor block were achieved within thirty five minutes of duration from the time of injection of local anaesthetic agent. Out of 20 cases in each group 7 cases (i.e. 35%) had complete block in group I, 16 cases (i.e. 80%) in group II and 15 cases (i.e. 75%) in group III.

Incomplete block was the one when any of the parameters other than pin prick could not be achieved within thirty five minutes of duration from the time of injection. Maximum number of incomplete block that is 13 out 20 cases (i.e. 65%) was seen in group I, next in group II 4 out of 20 cases (i.e. 20%) and lastly in group II that is 5 out of 20 cases (25%).

Block was declared failed when none of either motor or sensory parameters including pin prick could be achieved in duration more than thirty five minutes. Failed block was not seen in either of the groups.

Our observation are comparable with the findings of ***Radha Sukhani et al (1987)*** who found complete block in 54% cases in lignocaine carbonate (p^H 6.8) groups while 31% in lignocaine hydrochloride (p^H 6.5) group.

Schulte – Steinberg et al (1970) found 44% patients had a complete block when carbonated solution of lignocaine was used (p^H 6.5) but only 36.3% had such blockade when hydrochloride solution was used. (p^H 4).

Martin (1981) did not observe any significant difference in the quality of block while comparing lignocaine hydrochloride with hydrocarbonate in axillary brachial plexus block.

Chow et al. (1998) compared lidocaine with adrenaline solution (p^H 6.24) with alkalinized lidocaine with adrenaline solution (p^H 7.15) for axillary plexus block. He concluded that the difference in the overall success and adequacy of the axillary brachial plexus anaesthesia did not reach statistical significance between the two groups.

Duration of Block

In present study duration of sensory block was taken from the time of complete onset of sensory block till the patient respond to pin prick and the duration of motor block from the time of loss of finger movements till the patient started moving his fingers.

Mean duration of sensory block for group I cases 87.1 $\pm$ 17.10 minutes. Maximum increase in the mean duration of sensory block was seen with group II then with group III but the increase in their value was insignificant when compared with the value of group I. ($p > .05$).

Mean duration of motor block in group I cases was 107.4 $\pm$ 13.31 minutes. In group II cases after alkalinization of the local anaesthetic agent it increased to 117.55 $\pm$ 9.85 minutes and in group III cases to 115.9 $\pm$ 9.49 minutes. Increase in the values were found to be highly significant and significant respectively in comparison to group I.

From our study we can conclude that alkalinization of the lignocaine hydrochloride solution do improve the duration of motor block while having no effect on the duration of the sensory block. As the mean duration of sensory block was less in all the groups so it can be fairly said that the recovery of sensory fibers precede the recovery of motor fibers.

Results of our study are comparable with those of ***Alon P Winnie et al (1977)*** who in their study stated that as motor fibers are situated peripherally so they are last to recover in comparison to sensory fibers which are located in the center of the nerve fibers.

Radha Sukhani & Alon P Winnie (1987) in their study observed that the duration of anaesthesia was virtually identical with 1% lidocaine hydrochloride and 1.1% lidocaine carbonate solution.

PR Bromage. (1972) calculated shorting of duration of action of carbonated lidocaine by 12% from that of the hydrochloride solution in their study.

Ririe DG, Walker Fo, James RL, Butterworth J. (2000) performed median nerve blocks on 10 volunteers to compare the effects of 1% plain lidocaine with 1% lidocaine mixed with sodium bicarbonate 0.1 mmol/litre. Their data suggested that addition of bicarbonate to lidocaine for median nerve block significantly increased the rate of motor block.

Lastly ***Difazio CA, Carson H, Grosslight KR, (1986)*** compared the pH adjusted lidocaine solutions for epidural anaesthesia and demonstrated that the degree of improvement in time to onset and duration is directly related to the extent of difference in the pH of the solutions compared.

Vitals

In our study we looked for any changes in the mean values of pulse rate, respiratory rate, arterial pressure and oxygen saturation after performance of the block in all the groups. The preoperative values of the mean pulse rate, respiratory rate, mean arterial pressure and oxygen saturation were almost similar and within normal range among all the groups. After the performance of the block insignificant changes in the mean arterial pressure and oxygen saturation was observed in all the groups.

Regarding changes in the mean pulse rate values, after 15 minutes of injection of local anaesthetic drug highly significant fall was seen in group II with only significant and no change observed in group III and group I respectively. Maximum fall in the pulse rate was seen 45 minutes after the performance of block in all the groups. Later on in post-operative period the mean pulse rate value in all the three groups rise from their intra-operative mean values but was still below their pre-operative mean pulse rate values, with group II cases still showing highly significant fall.

As far as mean values of respiratory rate taken at various time interval are concerned in our study a highly significant fall in the respiratory rate from their preoperative value was observed in group II after 15 minutes of injection of local anaesthetic agent while group I and group III cases observed insignificant changes. After 45 minutes of the performance of block highly significant and more significant fall in the mean respiratory rates values was noticed by group II, group III and group I cases respectively. In post operative period significant fall in the respiratory rate from their pre-operative value was noticed in group II cases only while group I and group III cases had insignificant changes.

Complication

Out of 60 cases in our study, arterial puncture which is a common complication was encountered in 5 cases. Two cases each in group I and group II and only one case in group III.We did not encounter any case of pneumothorax, phrenic N. palsy and toxicity of local anaesthetic agent during our study.

N Harley and J Gjessing (1969) reported high incidence of arterial puncture in their study.

Regarding incidence of other complications ***De Jong (1961)*** has reported 2.5% incidence of pneumothorax in his study. ***Schulte Steinberg et al (1970)*** in the comparative study of lignocaine hydrochloride and lignocaine hydrocarbonate observed one case of phrenic nerve palsy in a patient receiving carbonated solution. ***Moore (1961)*** found 40% - 60% incidence of phrenic nerve palsy when he used 50 ml of local anaesthetic solution for brachial plexus block. He also reported 70-90% cases of stellate ganglion block in his case study.

None of the cases in our study showed signs of local anaesthetic toxicity. The significantly reduced incidence of these complication can be explained by the use of quite less volume of drug in our study. Cases were kept under observation for the period of 24 hours postoperatively No incidence of any complication was reported in the post-operative period.

CONCLUSION

CONCLUSION

From our study using the supraclavicular technique of brachial plexus block with lignocaine hydrochloride solution having three different p^H for surgery on the upper extremity, it was concluded that.

1. Raising the p^H of 2% lignocaine hydrochloride with adrenaline solution from 3.21 to 6.21, produced a definite reduction in the latency of sensory as well as motor block.

2. Further increase in the p^H of the 2% lignocaine hydrochloride with adrenaline solution to 6.67 did not confer any added advantage over the same local anaesthetic solution having p^H 6.21 in the reduction of the latency of sensory and motor block.

3. As per the duration of the block is concerned alkalinization of the 2% lignocaine hydrochloride with adrenaline solution improved the duration of motor block with no effect on the duration of sensory block.

4. Much improvement in the duration of motor block was seen with the solution having p^H 6.21 as compared to that having p^H 6.67.

5. In our study recovery of sensory fibre preceded the recovery of motor fibres.

6. Greater frequency of complete block was seen on increasing the p^H of 2%lignocaine hydrochloride to 6.21 from 3.21. No added advantage in the quality of block was seen on increasing the p^H further to 6.67.

7. No incidence of failed block was observed in either of the groups.

Therefore alkalinized lignocaine hydrochloride solution provides significant advantage over non-alkalinized lignocaine hydrochloride in term of quicker onset, duration and quality of block. This appears to be due to a more rapid rate of intraneural diffusion, production of ion trapping and resultant increase in the amount of active cation available at intraneural receptor site.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Akamatsu TJ, Siebold KH. The synergistic toxicity of local anaesthetics. ***Anaesthesiology*** 1967; 28 : 238

2. Alon Winnie, Kanchan P Patel. Pharmacokinetics of local anaesthetics during plexus block. ***Anaesthesia & Analgesia*** 1967; 56: 852.

3. Alon Winnie, Zia Durrani. Factors influencing distribution of local anaesthetics injected in the brachial plexus sheath. ***Anaesthesia & Analgesia*** 1979; 58: 225.

4. AV Gromley WP, Hill DA, Murray J M, Fee JP. The effect of alkalinisation of lignocaine on axillary plexus anaesthesia. ***Anaesthesia*** 1996; 51 (2) : 185-8

5. Atkinson RS, Rushman GB, Lee JA. A synopsis of anaesthesia (8^{th} edn) weight – PSG, Birstol. 1982.

6. AV Gandy CL. Anaesthesia for Duperytren's contracture. ***Hand clinics*** 1991; 7 (4) : 695-704

7. Bedder MD, Kozody R, Craig DB. Comparison of bupivacaine and alkalinized bupivacaine in brachial plexus anaesthesia. ***Anaesthesia & Analgesia.*** 1988; 67 : 48-52.

8. Benhaumou D, Labaille T, Bonhonome L, Perrachon N. ***Regional Anaesthesia*** 1989; 14:240.

9. Bokesch PM, Raymond SA, Strichartz GR. Dependence of lidocaine potency on pH & PCO_2. ***Anaesth. Analg.*** 1987; 66:9.

10. Bromage PR. Improved brachial plexus blockade with the hydrochloride and carbonated lignocaine. ***Anaesthesiology*** 1972; 36 : 479-87.

11. Burney R G, Difazio CA, Foster JA. Effects of pH on protein binding of lignocaine. ***Anaesth. Analg.*** 1980; 57 : 478.

12. Berman S, Rosenquist RW, Finucane BT. Alkalinization of mepivacaine for axillary brachial plexus block. ***Can. J. Anaesth*** 1998; 35 (3) :593-5.

13. Brown D T, Beamish D, Wildsmith JAW. Allergic reaction to an amide local anaesthetic. ***British. J. Anaesthesia.*** 1981; 33 : 435-437

14. Bromage PR, Burfoot MF, Crowell DE, Truant AP. Quality of epidural blockade. III : carbonated local anaesthetic solutions. ***British. J. Anaesth*** 1967; 39 : 197-208.

15. Caldwell PC. Studies on the internal pH of large muscle and nerve fibres. ***Journal of physiology.*** 1958; 192 : 22.

16. Capogna C, Celleno D, Laudano D, Giunata F. Alkalinization of local anaesthetics, which block, which anaesthetic. ***Regional Anaesthesia*** 1995; 20 : 369.

17. Catchlove, Richard F H., The influence of CO_2 & pH on local anaesthetic actions. ***J. Pharmacol. Exp. Ther.*** 1972; 181 : 298.

18. Catchlove, Richard FH. Potentiation of two different local anaesthetics by
Carbon dioxide. ***British. J. Anaesthesia*** 1973; 45 : 471-4.

19. Chow MHY, Chan WY. Alkalinization of lidocaine does not hasten the onset of axillary brachial plexus block. ***Anaesthesia Analg*** 1998: 86 : 556-8.

20. Defazio CA, Carron H, Gross light KR, et al. Comparison of pH adjusted lignocaine solution for epidural anaesthesia ***Anaesthesia. Analg.*** 1986; 65:760.

21. Donchin Y, Ramu A, Olshwong, Neiman Z & Magora F. Effect of sodium bicarbonate on the kinetics of bupivacaine in I.V. regional anaesthesia in dog. ***British. J. Anaesthesia,*** 1980; 52 : 969.

22. Gissen A, Covino BG, Gregus J. The differential senstivities of mammalian nerve fibres to local anaesthetic agents. ***Anaesthesiology*** 1980; 53 :467.

23. Gissen AJ, Cavino BG. Differential sensitivity of fast and slow fibres in

    mammalian nerve. Effect of carbonation of local anaesthetics ***Reg. Anaesth.*** 1985; 10 : 68.

24. Galindo A. p^H adjusted local anaesthetics. Clinical experience. ***Reg. Anaesth.*** 1983; 8 : 35-6

25. Harley N, Gjessing I. A critical assessment of supraclavicular brachial plexus block. ***Anaesthesia*** 1969; 24:564.

26. Helgier M. Alkalinization of bupivacaine for brachial plexus block ***Reg. Anaesth.*** 1985; 10 : 59.

27. Hille B. The pH-dependent rate of action of local anaesthetics on the node of ranvier. ***J. Gen. Physiol.*** 1977; 69 : 475.

28. Ikuta PT, Vasiraddy AR, Raza SM, Winnie AP, Duraniz, Masters RW. pH adjustment schedule for amide local anaesthetics. ***Regional Anaesthesia*** 1989; 14 : 229-35.

29. Lim EK, Pereira R. Brachial plexus injury following brachial plexus block. ***Anaesthesia*** 1984; 34:691.

30. Macintosh RR, Mushin W. Local Analgesia; Brachial plexus. 3rd edit. Edinburgh, E. & S. Livingstone.

31. Martin R, Beauregard L, Lamarche Y, Tetrault JP. Comparison of lidocaine hydrocarbonate, lidocaine hydrochloride and mepivacainc in axillary block. ***Can. J. Anaesth.*** 1987; 34:6.

32. Mcclure J H, Scott DB. Comparison of bupivacaine hydrochloride and bupivacaine carbonate in brachial plexus block by the interscalene technique ***British. J. Anaesth.*** 1981; 53 : 523.

33. Mcleskey CH. p^H of local anaesthetic solutions. ***Anaesth. Analgesia*** 1980; 59:892.

34. Moore DC. Regional block 3rd edn. Ch. 27. Spingfield, Illinosis, C.G. Thomas.

35. Moore DC. The pH of local anaesthetic solutions. ***Anaesthesia – Analgesia*** 1981; 60 : 833.

36. Mc Caughey. W. 1992 Adverse effects of local anaesthetic Drug safety 7 (3) : 178-189

37. Narahashi T, Frazier DT, Yamada M. The site of action and active form of local anaesthetics. ***J. Pharmacol exp. ther.*** 1970; 171: 32.

38. Nelson L C, Joel Akaplan. A rapid onset, long acting regional anaesthetic technique. ***Anaesthesiology*** 1974; 41:509.

39. Quinlan JJ, Oleksey K, Murphy FL. Alkalinization of mepivacaine for axillary block. ***Anaesth. - Analgesia*** 1992; 74 (3) : 371.

40. Ritechie JM, Ritchie B, Greengard P. The effect of the nerve sheath on the action of local anaesthetics. ***J. Pharmacol Exp. Ther*** 1965; 150 : 160.

41. Roberts JE, Macleod BA, Hollands RH. Improved peribulbar anaesthesia with alkalinazation and hyaluronidase. ***Canadian J.Anaesth.*** 1993; 40 (9) : 835.

42. Rosen blatt RM, Fung DL. Mechanism of action for dextron prolonging regional anaesthesia. ***Regional Anaesthesia*** 1980; 5 (2) :3-5.

43. Reiz S, Nath S. Cardiotoxicity of local anaesthetic agents. ***British Journal of Anaesthesia*** 1986; 58 : 736-745.

44. Ririe DG, Wakler FO, James RL, Butterworth J. Effect of alkalinization of lidocaine on median nerve block. ***British. J. Anaesth.*** 2000; 84 (2) : 163-8.

45. Ritchie JM, Ritchie BR. Local anaesthetics : Effect of pH on activity. ***Science*** 1968; 162 : 1394-5

46. Sadove, Wyantg M, Gittelson, Kretchmer. Classification and management of reaction to local anaesthetic agents. ***Journal of the American Medical Association.*** 1952; 148: 17.

47. Schulte-steinberg, Schutt L. Carbon dioxide salts of lignocaine in brachial plexus block. ***Anaesthesia.*** 1970; 25:191.

48. Seeman P. Anaesthetics and pressure reversal of anaesthesia. Expansion and recompression of membrane proteins, lipids and water. ***Anaesthesiology.*** 1977; 47:1.

49. Strichartz C. Molecular mechanism of nerve block of local anaesthetics. ***Anaesthesiology.*** 1976; 45 : 421.

50. Sukhani Radha, Alon P Winnie. Clinical phamacokinetics of carbonated local anaesthetics II: interscalene brachial plexus block model. ***Anaesth. Analgesia.*** 1987; 66 :1245.

51. Sukhani Radha, Alon P Winnie. Clinical Pharmaco kinetics of carbonated local anaesthetics I: interscalene brachial plexus block model. ***Anaesth. Analgesia*** 1987; 66 : 739.

52. Sukhani Radha and Winnie A P. Clinical pharmacokineties of carbonated local anesthetics I : subclavian perivascular brachial block model. ***Anaesth. Analg*** 1987; 66 : 739-45.

53. Tetzlaff JE, Walsh M, Yoon HJ. Alkalinization of mepivacaine decreases the latency to onset of axillary brachial plexus block. ***Anaesthesiology.*** 1994; 21(5) : 165.

54. Tetzlaff JE, Walsh M, Yoon HJ. pH adjustment of mepivacaine decreases the incidence of tourniquet pain during axillary brachial plexus anaesthesia. ***Anaesthesiology.*** 1993; 42 (2) :133.

55. Tetzlaft JE, Yoon HJ, Brems J, Javorsky T. Alkalinization of mepivacaine

improves the quality of motor block associated with interscalene brachial plexus anaesthesia for shoulder surgery. ***Regional Anaesthesia*** 1995; 20 (2) : 128.

56. Tuker GT, Mather LE. Pharmacokinetics of local anaesthetic agents. ***British. J. Anaesth.*** 1975; 47 : 213.

57. Verborgh C, Claeys M.A, Camu F. Onset of epidural blockade after plain or alkalinized 0.5% bupivacaine. ***Anaesth-Analgesia.*** 1991;73 (4) : 401.

58. Walker RT. Hypersensitivity reaction to local anaesthetic. ***Anaesth-Analgesia.*** 1971;130 : 2.

59. Wenker, Nolte. Brachial plexus block for local anaesthetic evaluation. ***British. J. Anaesthesia.*** 1974; 47 : 301.

60. Wong Kin, Gary R, Strichartz, Stephen A, Raymond. On the mechanism of potentiation of local anaesthetics by bicarbonate buffer: Drug-structure- activity studies on isolated peripheral nerve. ***Anaesthesia and Analgesia*** 1993; 76 : 131.

61. Wildsmith JAW, Gissen AJ, Cregns J, Covino B. Differential nerve blocking activity of amino-ester local anaesthetics. ***British. J. Anaesth*** 1985; 57 : 612-20.

62. Winnie AP, Tay C, Patel KP, Rammurthy S, Durrani Z. Pharmaco kinetics of local anaesthetics during plexus blocks. ***Anaesth. Analg*** 1977; 56 : 852-61.

63. Yamano Y. Safe method of supraclavicular brachial plexus anaesthesia. ***Arch Orthop Trauma Surg.*** 1983; 102 : 92.

APPENDIX

WORKING PROFORMA

DEPTT. OF ANAESTHESIOLOGY, M.L.B. MEDICAL COLLEGE, JHANSI

Name :

Age/Sex :

M.R.D. No. :

Drug used : Group

p^H of the Group :

Time of Performing Block :

Diagnosis :

Surgical Procedure

Duration of surgery

Vitals	Pre-operative	Intra-operative		Post operative
		15 Min	45 Min	
Pulse Rate				
Resp. Rate				
Blood Pressure				
SpO_2				

ONSET OF BLOCK

Onset of Sensory Block

Sensory Parameters			
Temp Mts	Touch Mts	Pin Prick Mts	Pressure Mts

Onset of Motor Block

Motor Parameters	
Paresis	Paralysis
Mts	***Mts***

QUALITY OF BLOCK

Complete: Incomplete : Failed :

DURATION OF BLOCK

Minutes

Duration of sensory block :

Duration of motor block :

COMPLICATIONS IF ANY

ABBREVIATIONS

ASA	:	American Society of Anaesthesiologist
%	:	Percentage
ml	:	Milliliter
wt/vol		Weight/Volume
sec	:	Second
cm	:	Centimeter
mv	:	Millivolt
pka	:	Dissociation Constant
hrs	:	Hours
mg/kg	:	Milligram/Kilogram
MAC	:	Minimum alveolar concentration
ug/ml	:	Microgram/Milliliter
mg	:	Milligram
S.D.	:	Standard Deviation
mmHg	:	Millimeter of Mercury
mts	:	Minutes